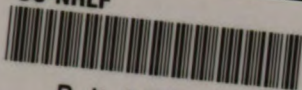
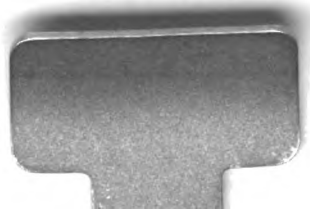


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AN INTRODUCTION TO CLINICAL PERIMETRY

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For General Practitioners and Students

By H. M. TRAQUAIR, M.D., F.R.C.S. (Ed.)

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AN INTRODUCTION
TO
**CLINICAL
PERIMETRY**

BY
H. M. TRAQUAIR, M.D., F.R.C.S. Ed.

CONSULTING OPHTHALMIC SURGEON, ROYAL INFIRMARY, EDINBURGH.
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WITH A FOREWORD BY
NORMAN M. DOTT, M.B., Ch.B., F.R.C.S. Ed.

SIXTH EDITION REVISED AND ENLARGED

WITH 257 ILLUSTRATIONS AND 5 COLOURED PLATES

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FOREWORD

DR. TRAQUAIR has honoured me by asking me to write a brief appreciation of perimetry from the standpoint of neurologist and neuro-surgeon by way of a foreword to this volume. With these terms of reference it would be out of order for me to laud the author and his work as I would in other circumstances. The volume places at the disposal of the medical profession an eminently practical guide to the technique of perimetry, rendering its practice attainable to all who will apply themselves to it. It lays before us in graphic form the author's rich store of personal observations carefully selected and condensed. It gives us the benefit of his ripe experience of interpretation and evaluation of pathological alterations of the visual fields. The author's status in the development of scientific and practical perimetry requires no comment by me.

It had long been recognised among neurologists that lesions of the brain might cause changes in the fields of vision and that the latter were of diagnostic value. Among neurologists, Cushing appears to have been the first to appreciate fully the value of and to insist upon routine quantitative perimetry in the clinical investigation of brain lesions. Cushing and Walker's contributions to visual field changes associated with brain tumour are recognised classics. This valuable material was gathered by routine perimetry of all cases of suspected brain lesions.

As I am fortunate to be a pupil both of Traquair and of Cushing my appreciation of the value of perimetry will hardly be doubted !

The author of this volume has often remarked in the investigation of brain disease that the function of vision is equally deserving of study as are those of motion and sensation. He has drawn attention to the wide extent of the intracranial visual pathways and their consequent liability to involvement in many cases of intracranial disease. He has demonstrated that in quantitative perimetry we have a relatively exact method of examination which compares favourably with the means at our disposal for the investigation of the other senses and of motor, intellectual and psychic functions. He teaches that the perimetric finding is but one of the many data to be ascertained and evaluated in composing a comprehensive picture of the underlying disease process. In practice I find these conceptions so important to the practical diagnosis of brain disease, the proper direction of brain surgery, and the adequate assessment and control of the results of treatment, that perimetry is a daily routine in the neuro-surgical department.

It is obvious that such means of examination as are generally useful to him must become an integral part of the neurologist's routine of investigation. Methods of examination, which, when first introduced, are regarded as "special examinations," are ultimately absorbed into ordinary clinical practice. The ophthalmoscope is a great gift from ophthalmology to neurology. At first an instrument peculiar to the oculist, it is now familiar to the neurologist. The perimeter is, I believe, an even greater gift from the sister science to neurology. Although it was offered earlier than the ophthalmoscope yet neurologists in general have been ungratefully slow in accepting it. If he who proposes to diagnose diseases of the brain is not as familiar with the perimeter and screen

and with the interpretation of pathological changes in the fields of vision as he is with the fundus oculi, his equipment is seriously incomplete.

In clinical neurological practice it is inconvenient and impractical to call in the ophthalmologic specialist for the daily routine examination of the fields of vision which the adequate study of brain functions requires. Such a method can only lead to omission of perimetry where it would prove of vital importance. Every neurologist, therefore, will be especially grateful for this helpful volume. Although the neurologist should conduct his own routine perimetry, yet occasion will frequently arise for him to seek the aid of his ophthalmologic colleague. The oculist's knowledge of the field changes associated with primary ocular and optic nerve diseases must always surpass that of the neurologist. His aid in the differential diagnosis of simulating nervous diseases will be required. Reciprocally the oculist seeks the aid of neurologist or neuro-surgeon in the cases of patients with intracranial lesions who often consult him primarily by reason of their ocular symptoms. The practice and knowledge of perimetry should be common ground to both branches of medicine.

As set forth in detail in this volume it will be appreciated that the site and form of a defect in the field of vision furnish clear evidence of the fibres involved as considered in a cross section of the visual pathway. These factors also furnish exact or approximate evidence of the level of the lesion as considered in relation to the longitudinal dimensions of the visual pathway. The character of the defect, its sharp or diffuse margin, and its behaviour as ascertained by repeated examinations, reflect the nature of the pathologic process in relation to its fixity, progression or retrogression. The direction of its extension, as from above or below the visual pathway, may also be ascertained. It is evident that information of this order is of the utmost importance to adequate diagnosis and proper direction of treatment. The perimetric information is to be duly aligned with other clinical facts, the history of symptoms, the presence or absence of other evidences of cerebral or spinal derangement, the presence or absence of signs of vascular or metabolic disease, with the age of the patient, etc. The time factor in relation to field changes is also important. The abrupt development of a gross sectoral field defect in one eye, associated with œdema and engorgement of the nerve head, suggests a vascular lesion of the optic nerve. The rapid development of an extensive central scotoma in the field of one or both eyes associated with œdema of the nerve head suggests an inflammatory focus in the optic nerve which may accompany, precede or follow acute disseminated encephalomyelitis. When papilloœdema precedes the development of a unilateral or, less frequently, a bilateral central scotoma, the most probable cause is increased intracranial pressure, with the later advent of direct intracranial compression of the optic nerve by frontal tumour or even by a distended third ventricle.

It is well to remember that a defect in the field of vision proves nothing more than a defect in conduction in a certain part of the visual organ or pathway. One must not strain its value beyond this limitation. In relation to the intracranial visual pathway no perimetric finding can be regarded as pathognomonic of a certain disease. In most cases associated circumstantial evidence will permit a reasonably accurate inference to be drawn. It may be proper, however, to operate on a patient upon perimetric evidence

alone. For example, if the perimetric picture points to a lesion at the under-surface of the anterior angle of the optic chiasma and if it is steadily progressive we may infer a slowly enlarging mass in this situation. Beyond this, in the absence of other clinical or radiological evidence, we cannot go. We may consider that a pituitary chromophobe adenoma is most probable but we cannot exclude a meningioma or aneurysm. Yet as the majority of slowly enlarging tumefactions here are susceptible of satisfactory surgical treatment it will be proper to operate.

At the other end of the scale we may have to deal with a patient with every evidence of increasing intracranial pressure urgently requiring relief. In such a case it may happen that a searching neurological and radiological study fails to locate the disease process but that careful perimetry may yield definite information and direct treatment.

One must not overlook the value of definite proof of the absence of involvement of the visual pathway. In order that such proof may be regarded as definite it is necessary that careful quantitative perimetry as described in this volume should be carried out. Allowance may be required for the effects of papilloedema and its consequential optic atrophy, as contrasted with direct implication of the visual pathway by the disease. In this way it is obviously possible to exclude a very considerable part of the brain as a possible site of the disease process, and thus to narrow down the search for the lesion.

Dr. Traquair has emphasised the subjective nature of the perimetric examination. While it lends itself to greater accuracy than most other methods of clinical examination it is not a method of mathematical precision. These considerations apply with particular force to many patients suffering from brain disease. These patients are often seriously ill; their capacity for concentration on the test may be impaired; they are often very rapidly fatigued by the examination. Their responses to the test become increasingly unreliable and inaccurate in proportion as these symptoms prevail. The neurological perimetrist must learn to assess the value to be placed on the patients' responses in these circumstances. He must realise that, within reasonable limits, a more accurate idea of the visual field may be obtained by sacrificing time-consuming, meticulous precision to speed of examination. For he must avoid the very gross inaccuracies which necessarily accompany undue fatigue of the patient. It is desirable in dealing with such cases that the perimetrist should learn to memorise the whole of an isopter so that he can obtain its outline quickly without stopping to chart or mark it. The patient is then put at ease with closed eyes, while the finding is charted. Thus the patient is required to concentrate for a minimum length of time and is frequently rested. Often, in spite of these precautions, disabling fatigue supervenes before all the necessary information has been obtained, and it is necessary to postpone further examination for minutes or hours according to circumstances. For rapid perimetry the wide segment perimeter of the author or that of Walker (Fig. 10) is advantageous. In brain tumour cases there is often an element of urgency in securing diagnostic data. Artificial lighting is advantageous for perimetry in these cases as it may be undesirable to postpone examination in order to secure satisfactory daylight conditions. In some cases where drowsiness and apathy are very marked one may obtain more reliable information concerning gross defects of the visual fields by the confrontation method than by the use of perimeter or screen. The neuro-

logical perimetrist should be thoroughly familiar with this method. Especially he should learn to observe the patients' response objectively so that he may gain information in the absence of verbal replies, as in aphasic patients and in young children. While the examination of the inco-operative patient has been stressed in considering neurological perimetry it should be added that many patients with brain disease are normal in their capacity for perimetric examination. The greatest accuracy becomes especially important in the analysis of small central and paracentral defects.

Pathological changes in the visual fields are as important to the neurologist and neuro-surgeon as those of the knee jerk or plantar response or the fundus oculi. If they are not thoroughly familiar with the practice and interpretation of perimetry they are incompletely equipped for their work. Those who avail themselves fully of this means of examination have at their command one of the most important and most helpful means of investigating organic lesions of the nervous system.

NORMAN DOTT.

PREFACE TO THE SIXTH EDITION

Two Coloured Plates and two new illustrations have been added, and the text has been revised and amplified.

I am very grateful to Mr. Keith Lyle for allowing me to use his excellent coloured drawing of opaque nerve fibres.

Mr. Henry Kimpton has, as usual, taken every care, and spared no trouble.

H. M. T.

EDINBURGH, 1949.

PREFACE TO THE FIFTH EDITION

FOR the fifth edition the anatomical terminology has been brought into line with modern usage. For this and for much helpful criticism and for assistance in correcting the proofs I express my grateful thanks to Professor Brash of Edinburgh University. The text has been revised, ten new illustrations and sixteen references have been added.

I gratefully acknowledge the care and trouble taken by Mr. Henry Kimpton.

H. M. T.

EDINBURGH,
January, 1946.

EXTRACT FROM THE PREFACE TO THE FIRST EDITION

THIS book is an amplification of the Middlemore Essay for 1920 on "Perimetry (inclusive of Scotometry), Its Methods and Its Value to the Ophthalmic Surgeon." It does not attempt to deal exhaustively with perimetry, but only to introduce the reader to its essential principles considered in their clinical aspect.

The presentation of the subject has, therefore, been limited to what the clinician is likely to find useful. Incursions into the more academic aspects of visual response have been reduced to the lowest limit compatible with providing some kind of explanation for clinical findings.

In dealing with the methods of field testing, I have endeavoured to describe and explain a simple procedure which the experience of others as well as myself has shown to be both easy and satisfactory. No attempt has been made to review the numerous variations of method or of apparatus, often elaborate and costly, which have been advocated from time to time, not from any desire to depreciate their value, but because part of the object of this work is to urge that success in perimetry, as in many other forms of subjective examination, will not be attained by the use of the "best" or the "newest" instrument, but by the study and application of simple principles. The procedure described is that known as the quantitative method of perimetry which is essentially the method of Bjerrum, who discovered, more than thirty years ago, that he could obtain more information by using the back of his consulting-room door than he could from the ordinary perimeter. Originally directed to the study of glaucoma, the principle of Bjerrum's method has been extensively applied by Roenne, at the present time the chief exponent of quantitative perimetry, who has demonstrated its advantages in the examination of all conditions in which perimetry is useful, and who introduced the term "quantitative" to indicate its analytical nature. This form of field testing was introduced to English-speaking ophthalmologists in 1890 by Berry, who had previously himself been working along the same lines. Since then the quantitative method has been further developed, and its value, both as a means of diagnosis and of research, is becoming increasingly appreciated.

Not less essential than adequate examination is the correct interpretation of the results obtained. In this connection the anatomy of the visual path, the importance of which to the perimetrist can hardly be overestimated, has been emphasised, and some reference has been made to pathological conditions with a view to correlating these factors with alterations in function and assigning a rational significance to field changes. On these lines I have attempted to discuss the genesis of field changes and the indications which they afford.

In estimating the significance of alterations in the fields, especially in relation to diagnosis, stress has been laid throughout on the necessity of considering such changes as part only of the evidence upon which conclusions are to be based, though at the

same time, in order to avoid unduly enlarging the scope of the book, references to ophthalmoscopic and other signs have been almost entirely omitted.

Some of the fields illustrated have been designed to indicate the various degrees of visual acuity by differential shading, so that each field resembles a map of a mountain. The bulk of the illustrations are from my own collection ; a few have been borrowed from other sources.

I do not venture to expect that this book will not be found to contain much that is inaccurate and more that is immature. My hope is that it may in some degree stimulate interest in perimetry and in the study of the anatomy and pathology of the visual nerve path, not only among oculists, but also among physicians and surgeons.

Mr. Henry Kimpton has been most patient and considerate, and has done everything possible to meet my wishes.

H. M. T.

EDINBURGH,

April, 1927.

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CLINICAL PERIMETRY

PART I

THE FIELD OF VISION : PERIMETRIC INSTRUMENTS AND METHODS

INTRODUCTORY

THE field of vision is that portion of space in which objects are visible at the same moment during steady fixation of the gaze in one direction. With every movement of the eye the field, while remaining practically the same in area, changes its contents at its margins so that as the eye is nearly always moving slightly, and as the effects of visual impressions persist for longer than an instant, the extent of the field for practical purposes is somewhat larger than in the case of an actually stationary eye, except in such directions as it is limited by the contours of the orbit. Perimetry, or the measurement of the field, is concerned only with the field of the stationary eye, that is, of the eye when its visual axis is directed as exactly and steadily as possible towards a fixed point. The distinctness with which objects in the field are perceived depends on various physiological factors of which the most important is proximity to the visual axis. The surface of the retina, the peripheral receptive organ, receives a minified and inverted image of the field, just as a photographic plate receives an image of a landscape. The fibres of the conducting nerve path are arranged side by side so as to preserve, with certain qualifications, the arrangement of the retina. Thus a transverse section of the path at any level repeats the retina, and, therefore, the field, and the same applies to the receptive centre, the visual cortex or cortical retina. The visual nerve mechanism may be compared to a telephone consisting of receiver, wire and station. Faults may be situated at any point, and may be of various kinds. The object of perimetry is to ascertain the presence, position and nature of faults in the visual nerve apparatus. Since the field corresponds to a cross-section of the nerve-path, a defect in the field will, if properly interpreted, afford some guidance as to the situation and nature of the causal lesion, whether this is in either of the end stations or in the conducting fibres.

Before we can practise perimetry, or "take the field" with success, it is necessary to have clear ideas as to what we are undertaking, and some kind of mental picture is essential. Any attempt at a full exposition of the field, with its physics and its psychology, is out of place in this connection. We must aim at some simple image which will illustrate the essential features in such a way as to assist the clinician.

To define the field of vision as the projection into space of the light rays which fall upon the sensitive retina during fixation of the eye may be accurate, as far as it goes, but is clinically sterile. It will be found more useful to regard it as a portion of an immense hollow sphere upon the inner surface of which is spread a panoramic picture of external objects showing the central feature depicted with minute detail and vivid colouring, while objects at increasing distances from the centre are indicated with correspondingly diminished clearness and duller hues. But the most helpful mental

picture of the visual field is obtained when we regard it from the standpoint of visual acuity. We may imagine the field as an island* of vision surrounded by a sea of blindness (Figs. 1, 2, 3). The coast-line is somewhat ovoid in shape, and rises steeply so that the island is surrounded by cliffs vertical at one side, sharply sloping at the other. Above the cliffs is a sloping plateau which rises more rapidly again towards the somewhat eccentrically situated summit. This is crowned by a sharp pinnacle whose sides curve steeply upwards from a narrow base. To one side of this point is a pit (the blind spot) with sides almost vertical at first, but soon becoming perpendicular, which extends down to the level of the surrounding sea. To an observer situated in the air above the pinnacle a panoramic view of the whole island is presented. On the shore only large objects can be seen and colours cannot be distinguished. Immediately within the coast-line along the top of the cliffs smaller objects are visible and colours can be recognised if in large enough patches, and as the neighbourhood of the summit is approached smaller and smaller objects become apparent until at the apex of the pinnacle the most minute details can be detected. Imagine the surface of such a hill, not stationary, but subject to slight fluctuations in height, and we obtain a glimpse of the normal field of vision, and we realise that the problem of perimetry is the survey of this surface.

If we choose to exercise our imagination still further and picture to ourselves this hill under abnormal conditions, we must think of it as distorted or partially destroyed by subsidence. Depressions of every variety may occur on its surface, of all shapes, sizes and depths, with straight, curved or irregular margins, with sloping or steep sides, isolated or extending to the shore or to the pit, or partly or wholly involving the central pinnacle.

Until comparatively recently, until the introduction of the quantitative method by Bjerrum and Roenne, the survey of this hill was largely neglected, and perimetry meant little more than the outlining of its coast-line and of deep depressions, frequently by rough and superficial methods. Modern perimetry is more ambitious, and, therefore, more exacting. It demands a thorough exploration of the whole area, an orographical survey as it were, with all differences in level shown by contour lines, neglecting no depression be it ever so restricted or so shallow. It will be seen that perimetry is a specialised study of one aspect of the symptomatology of vision comparable in many ways to the study of other kinds of disordered sensation. It is a study, adapted to clinical purposes, of the reception and conduction of impulses in the visual nerve apparatus and of the results of impairment of this apparatus. Its aim is to ascertain and distinguish, within certain limits, various forms of deviation from normal vision and to correlate each alteration as far as possible with a definite anatomical site of interference and a definite causation, and thus, in harmonious association with other methods, to contribute its quota to the production of a clear and complete clinical picture.

Perimetry has therefore two sides, the technical and the interpretative. For the former the perimetrist requires training and experience in practical methods of examina-

* The idea of the field of vision as an island in a sea of blindness derives originally from Euclid and from Heliodorus, who thought the field was coneshaped with a circular base, outside of which nothing could be seen. (Lloyd, 259.)

tion, and an acquaintance with the chief physical and physiological factors upon which these methods are based ; in respect of the latter, his knowledge of the anatomy of the visual nerve path and of the various ways in which different pathological conditions may affect it, cannot be too wide or profound. Further, since the examination is subjective and carried out by question and answer, due recognition must be given to purely psychical conditions which may influence the patient's responses.

It must, therefore, be emphasised that perimetry cannot be relegated to untrained persons, such as nurses or orthoptists, if satisfactory results are desired.

CHAPTER 1

THE NORMAL FIELD OF VISION

WE have seen that the field may be regarded as a hill of vision surrounded by a sea of blindness. For purposes of description, we must consider this hill as shown upon a map or chart by contour lines in the usual way (Figs. 1, 2). The centre point of

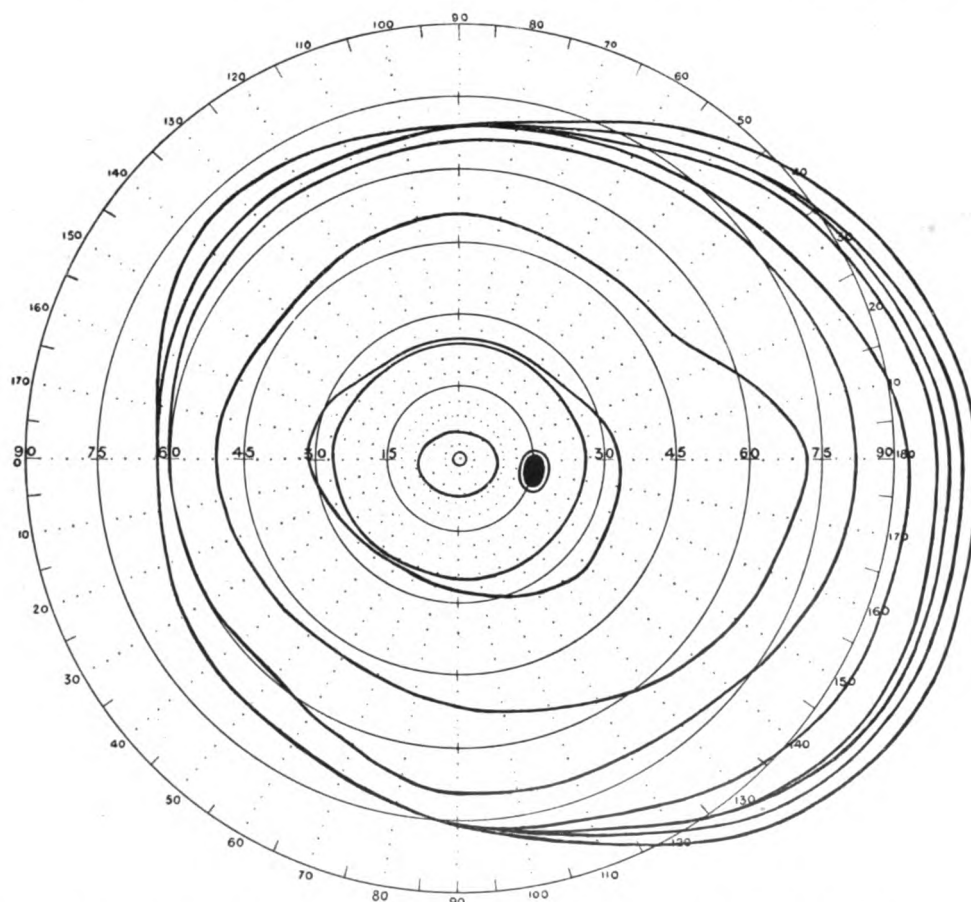


FIG. 1.—CHART OF THE VISUAL FIELD OF THE RIGHT EYE, SHOWING THE ISOPTERS FROM THE PERIPHERY INWARDS TO THE CENTRE FOR $\frac{100}{1000}$, $\frac{200}{1000}$, $\frac{300}{1000}$, $\frac{400}{1000}$, $\frac{500}{1000}$, $\frac{600}{1000}$, $\frac{700}{1000}$, $\frac{800}{1000}$, $\frac{900}{1000}$, $\frac{1000}{1000}$, $\frac{2000}{2000}$, $\frac{3000}{2000}$, $\frac{4000}{2000}$, $\frac{5000}{2000}$, $\frac{6000}{2000}$, $\frac{7000}{2000}$, $\frac{8000}{2000}$, $\frac{9000}{2000}$, $\frac{10000}{2000}$, $\frac{100}{4000}$, $\frac{200}{4000}$, $\frac{300}{4000}$, $\frac{400}{4000}$, $\frac{500}{4000}$, $\frac{600}{4000}$, $\frac{700}{4000}$, $\frac{800}{4000}$, $\frac{900}{4000}$, $\frac{1000}{4000}$, $\frac{2000}{4000}$, $\frac{3000}{4000}$, $\frac{4000}{4000}$, $\frac{5000}{4000}$, $\frac{6000}{4000}$, $\frac{7000}{4000}$, $\frac{8000}{4000}$, $\frac{9000}{4000}$, $\frac{10000}{4000}$, $\frac{100}{6000}$, $\frac{200}{6000}$, $\frac{300}{6000}$, $\frac{400}{6000}$, $\frac{500}{6000}$, $\frac{600}{6000}$, $\frac{700}{6000}$, $\frac{800}{6000}$, $\frac{900}{6000}$, $\frac{1000}{6000}$, $\frac{2000}{6000}$, $\frac{3000}{6000}$, $\frac{4000}{6000}$, $\frac{5000}{6000}$, $\frac{6000}{6000}$, $\frac{7000}{6000}$, $\frac{8000}{6000}$, $\frac{9000}{6000}$, $\frac{10000}{6000}$, $\frac{100}{8000}$, $\frac{200}{8000}$, $\frac{300}{8000}$, $\frac{400}{8000}$, $\frac{500}{8000}$, $\frac{600}{8000}$, $\frac{700}{8000}$, $\frac{800}{8000}$, $\frac{900}{8000}$, $\frac{1000}{8000}$, $\frac{2000}{8000}$, $\frac{3000}{8000}$, $\frac{4000}{8000}$, $\frac{5000}{8000}$, $\frac{6000}{8000}$, $\frac{7000}{8000}$, $\frac{8000}{8000}$, $\frac{9000}{8000}$, $\frac{10000}{8000}$, $\frac{100}{10000}$, $\frac{200}{10000}$, $\frac{300}{10000}$, $\frac{400}{10000}$, $\frac{500}{10000}$, $\frac{600}{10000}$, $\frac{700}{10000}$, $\frac{800}{10000}$, $\frac{900}{10000}$, $\frac{1000}{10000}$, $\frac{2000}{10000}$, $\frac{3000}{10000}$, $\frac{4000}{10000}$, $\frac{5000}{10000}$, $\frac{6000}{10000}$, $\frac{7000}{10000}$, $\frac{8000}{10000}$, $\frac{9000}{10000}$, $\frac{10000}{10000}$.

The numerator of each fraction represents the diameter of the test object in millimetres, the denominator the distance from the patient. It will be seen that as the test object is reduced in size the field becomes smaller, slowly at the periphery, but rapidly towards the centre. On the nasal side the larger test objects all give the same extent of field, so that the nasal edge of the field is perpendicular, the temporal edge being steeply sloping. The geometrical centre of the field is about 20° to the temporal side of the visual axis or physiological centre.

the chart corresponds to the visual axis, the object directly regarded, the fixation point or centrum. The area of the map is measured in degrees from this point, round which the coast-line of the island or peripheral boundary of the field is indicated by the most peripheral or lowest contour line.

These lines are termed isopters. The position of any isopter is determined by the size of the visual angle subtended by the test object to which it corresponds. Thus if an object of 160 mm. diameter is used at 1,000 mm. distance, the angle subtended is $\frac{160}{1000} \times \frac{180^\circ}{\pi}$, and the isopter is the isopter for $\frac{160}{1000}$ and the contained field is the field for $\frac{160}{1000}$ (see p. 9). The isopters, as in the case of contour lines on a plan of a mountain, are peripheral or low and internal or high.

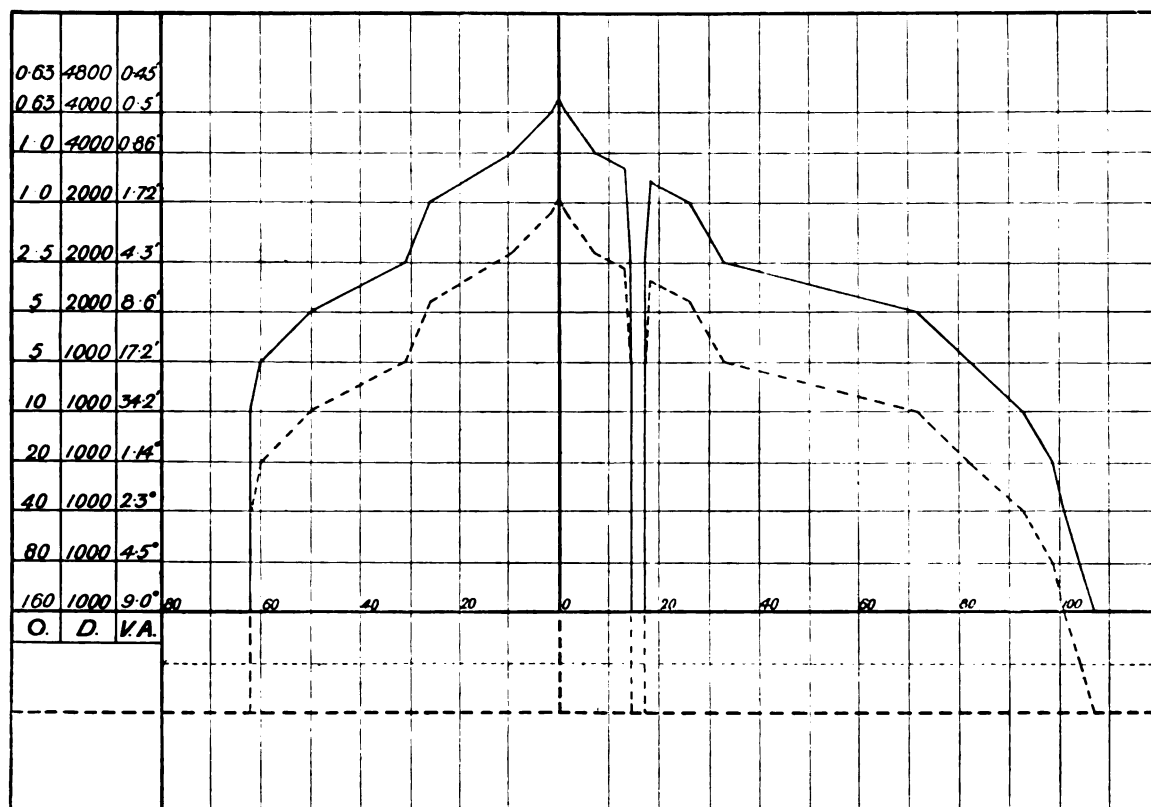


FIG. 2.—THE FIELD OF VISION REGARDED AS A HILL SEEN IN SECTION.

Modified from Roenne (338). The horizontal meridian is shown. Cf. Fig. 1. The continuous horizontal base line indicates the extent of the field in degrees, the vertical lines the visual acuity: O the diameter of the test-object in millimetres, D its distance from the eye in millimetres, and V.A. the visual angle subtended at the nodal point. Beginning with an angle of 9°, the visual angle is halved for each successive isopter; a modification is introduced by the substitution of $\frac{1}{2} \times 0.05$, and $\frac{1}{4} \times 0.05$ for the original figures (Roenne) of $\frac{1}{4} \times 0.05$ and $\frac{1}{8} \times 0.05$. The interrupted line shows the alteration produced by an imaginary *uniform* depression of the acuity over the whole field. The blind spot appears as a pit with crateriform mouth. It is shown a little wider than it should be, as if its full width lay on the horizontal meridian instead of slightly below it. This chart is intended to illustrate a clinical rather than a mathematical conception of the composition of the visual field in terms of relative visual acuteness; it is at least approximately accurate.

The field is usually charted as seen by the individual whose field is represented. Thus the field of the right eye is placed upon the right side of the chart with its temporal or right side towards the right, and its nasal or left side towards the left, and the field of the left eye is placed upon the left side of the chart with its temporal side to the left.

It is unfortunate that the term "normal," as applied to the field, does not yet connote features or limitations generally understood and agreed upon. Thus the field

is sometimes said to be normal, although all that has been ascertained is that its peripheral limit is normally situated. For clinical purposes, with which alone we are here concerned, we may regard a field as normal when the extent of its boundaries and its sensitiveness over its whole area attain the standard found in the average healthy individual by adequate and at the same time practical methods of examination. What this standard really amounts to will naturally be better understood when these methods have been discussed.

In this sense the normal field may be regarded as consisting of a central area extending to 25° in every direction from the fixation point, and a peripheral zone which includes

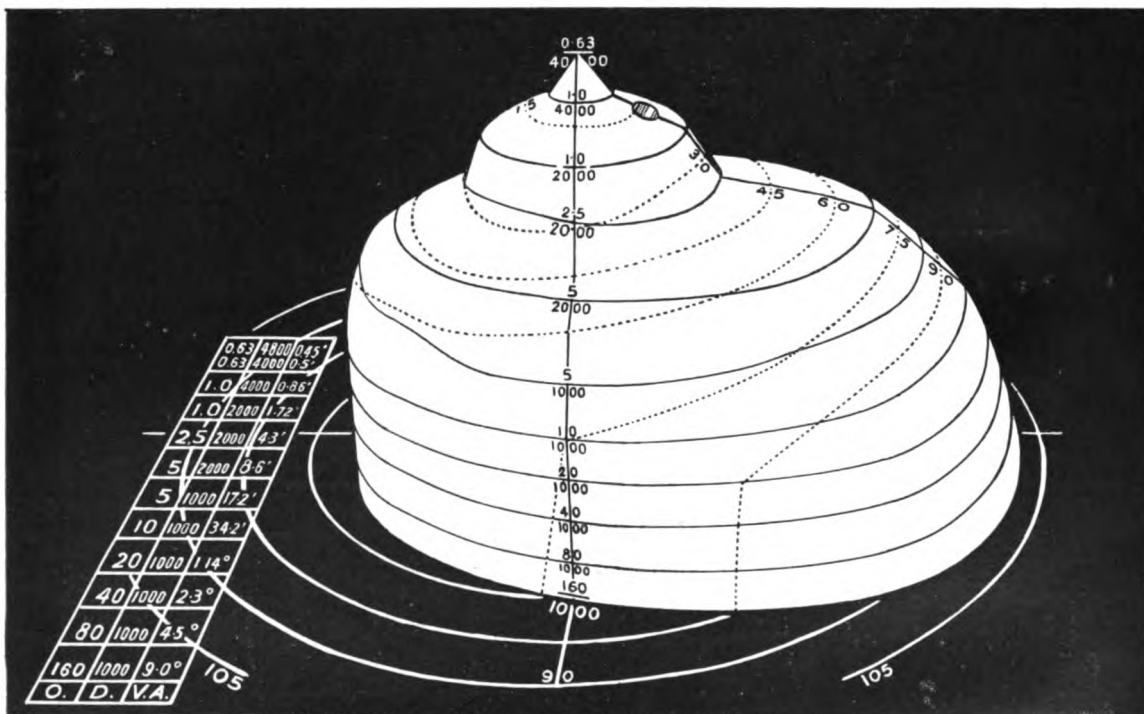


FIG. 3.—MODEL OF THE FIELD OF VISION FROM THE MEASUREMENTS GIVEN IN FIGS. 1 AND 2.

Note the steep nasal and more sloping temporal periphery, and the more gentle slope on both sides immediately within the periphery.

the remainder. In the centre, closely surrounding the fixation point, is the fixation area which corresponds to the projection of the macula.

A line passing perpendicularly through the fixation point constitutes the vertical meridian of the field and divides it into two unequal parts, a larger temporal and a smaller nasal "half-field" or field. A horizontal line through the fixation point divides each of these into upper and lower quadrants. The peripheral boundary is ordinarily determined nasally and upwards to a certain extent by the configuration of the orbital margin, nose and upper lid, and by the position of the eyeball in the orbit. In the case of the average individual, the field extends to about 60° upwards, 60° nasally, 75° downwards and 100° or a little more temporally. In the lower nasal quadrant the margin frequently recedes to 50° or 45° on account of the nose.

This is often called the *relative* field, in contrast to the *absolute* or maximum field, which is obtained when the eye is fixed and the face turned during the examination of each meridian in such a way as to exclude the influence of the orbital margins. The upper lid is elevated if necessary. A close approximation to the absolute field may be obtained in cases of exophthalmos from goitre or orbital tumour (see also Malbran, 276).

The absolute field is somewhat larger than the relative field in those directions in which the latter is restricted by the physical features referred to, the difference depending on the configuration present in individual cases. It does not extend to more than about 70° in any direction upwards or to the nasal side.*

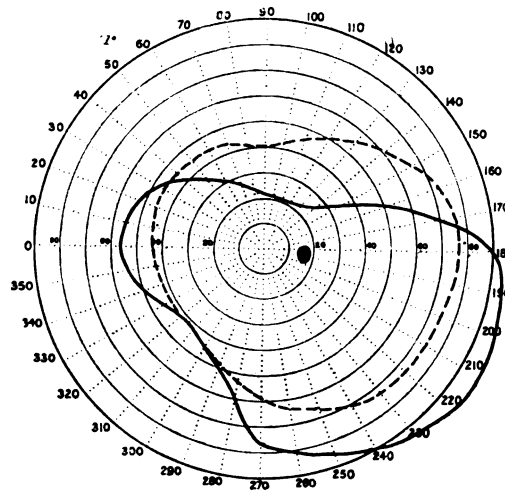


FIG. 4.—THE RELATIVE FIELD FOR $\frac{5}{30}$, SHOWING THE REDUCTION DUE TO A LARGE NOSE AND DROOPING UPPER LID.

The broken line shows the field for $\frac{5}{30}$ obtained by raising the lid, leaving the position of the face unaltered. The isopters coincide where the limitation is due to the nose.

The field of the two eyes together, or binocular field, is the combination of the right and left uniocular fields, which partly overlap, so that the nasal field of one eye covers the greater portion, the paired portion, of the temporal field of the other, leaving an outer crescentic uniocular area unpaired. This is called the temporal crescent or sometimes temporal "half-moon." The central portion common to both eyes is nearly circular with a diameter of about 120° , the temporal uniocular area extending to about 30° to 40° further on each side, so that the whole binocular field forms a rough horizontal oval extending to about 200° horizontally and 130° vertically. Within the paired part of the binocular field visual acuity is slightly greater than in the same area of either field separately. In the unpaired part of the binocular field I have found it by perimeter tests to be, if anything, a little less than in the same part of the uniocular field.†

The clinical perimetrist is mainly concerned with the uniocular relative field,

* In flat-nosed races (Chinese, Negroes) the nasal margin of the field shows no special extension.

Socrates was only partially correct in regarding his own flat nose as allowing the eyes "unobstructed vision." Xenophon. Banquet V.

† Appendix, Note I., p. 290.

though in some cases it is necessary to turn the face or lift up the upper lid of the patient in order to avoid error.

Within this area perception is much lower at the periphery than near the fixation point, towards which it gradually increases, though in the peripheral field moving objects are relatively easily detected.

Thus a stimulus which is only just strong enough to cause a visual impression in the central area will be invisible in the peripheral zone and the strength of a stimulus

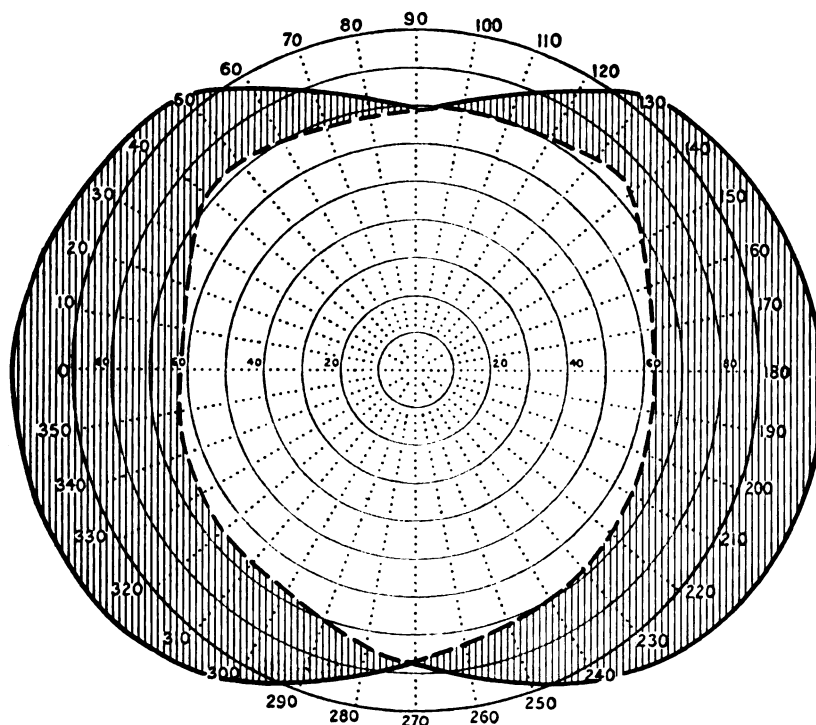


FIG. 5.—THE BINOCULAR FIELD, SHOWING THE CRESCENTIC UNPAIRED AREAS.

needful for its perception becomes gradually less as the fixation area is approached. In perimetry there are several methods by which the strength of the stimulus may be varied.

- (1) By using test-objects of different sizes.
- (2) By altering the distance at which the test-object is used.
- (3) By altering the colour of the object.
- (4) By altering the illumination.
- (5) By altering the background against which the object is seen.

In the first instance we may consider the field of vision as delimited by the use of white test-objects of varying sizes against a black background. By the use of a series of test-objects in this way, suitably graduated as to size, it is possible to ascertain the point of visibility for each size of object in a number of meridians, and thus to determine the position of the isopters and to show the slope of increasing visual acuity in the same way as altitudes are shown by contour lines on a map. Each isopter corresponds to a

certain size of object used at a certain distance, and as these factors may vary in relation to one another, the visual angle subtended by the object at the nodal point of the eye, and not merely its diameter, forms the proper basis of comparison. This angle is expressed in degrees by the fraction $\frac{\text{object}}{\text{distance}} \times \frac{180}{\pi}$. The constant $\frac{180}{\pi}$ or 57.3 is usually omitted, thus the extent of the field for an object of 1 mm. used at a distance of 330 mm. is referred to as the field for $\frac{1}{330}$, an expression which conveys more information than 10.4', to which it corresponds.* It is obvious that where the peripheral boundary of the field is determined by the nose or brow, *i.e.*, at the margin of the relative field, the image of a test-object brought centripetally into the field falls at once within the limit of the functioning retina, appearing to the subject "like the moon on the horizon" (Hess 176).

At such parts several isopters will coincide. A finding of this kind is not likely to be misinterpreted even by the inexperienced.

The outer boundary of the field constitutes the most peripheral isopter and can be marked out in an average healthy individual by using an angle of 30' ($\frac{3}{330}$) except on the temporal side, where a considerable increase in the angle shows the field to extend well beyond the 30' isopter. Figs. 1 and 2 and the following table show the isopters found by Roenne. With an angle of 9° ($\frac{180}{1000}$), Roenne found the field to reach 107° temporally,

Isopters in the Normal Field (Roenne 336)

Visual Angle.	Object (mm.).	Distance (mm.).	Out.	Out Down.	Down.	In Down.	In	In Up	Up	Out Up.	Reciprocal Value of Visual Angle.
9°	160	1,000	107	102	76	67	62	72	69	95	1
4.5°	80	1,000	104	99	76	67	62	72	69	91	2
2.25°	40	1,000	101	96	76	67	62	72	69	91	4
1.14°	20	1,000	99	96	76	67	62	72	69	88	8
34.2'	10	1,000	93	89	76	67	62	67	69	79	16
17.2'	5	1,000	82	72	69	59	60	64	66	75	32
8.6'	5	2,000	72	62	51	47	50	47	51	50	64
4.3'	2.5	2,000	33	34	27	25	31	25	24	25	128
2.1'	2.5	4,000	10	17	13	12	15	15	11	12	256
1.0'	1.25	4,000	10	9	8	7	6	6	4	6	512
0.5'	0.63	4,000	About 1° 20' Recognisable at the fixation point.								1,024
0.45'	0.63	4,800									1,236

It is evident from this table that the visual field has a very steep edge, especially on the nasal side. (See Figs. 1, 2, 3.)

* It is essential that the size of the object and its distance from the eye be given and not merely the visual angle, hence the method described above is the only satisfactory one. The reason is that under ordinary consulting-room or hospital conditions a slight variation may be found in the fields taken with the same visual angle at different distances in the normal eye, and in pathological conditions the difference may be more noticeable, when the object and distance vary—*e.g.*, $\frac{3}{330}$ does not always give the same field as $\frac{1}{330}$. Also in recording the conditions under which a field has been tested the fraction $\frac{O}{D}$ gives the size of the object and the distance at which it was used as well as the visual angle, whereas a statement of the visual angle alone gives no information whatever about the distance or the size of the object. See Appendix I., p. 289.

and 62° nasally, while an angle of $34'$ gave the same extent nasally, but only 93° temporally. Angles smaller than $30'$ give isopters lying within the periphery all round. The intervals between these boundaries are wider on the temporal than on the nasal side, so that as the visual angle is reduced the field becomes more circular in shape as well as smaller. With an angle of $1.7'$ the field is reduced to an approximate circle of about 26° , and with half that angle ($51''$) its extent is only about 8° , and so on, down to an angle of $27''$, which Roenne found to be seen only just at the fixation point.

The average normal positions of the isopters for definite visual angles with white test-objects can only be approximately established, and in many instances the observations hitherto published by various observers do not correspond. The nature of the subject indicates that only a limited degree of exactitude should be expected, and that conclusions must be based upon the examination of a large number of individuals under identical conditions. The main fact is, however, firmly established that the peripheral sensibility of the retina can be graphically expressed by isopters representing the extent of field corresponding to the visual angles subtended by the tests employed. This fact forms the basis of modern perimetry, and the term "quantitative" perimetry means perimetry based on the recognition of this principle.

The Field for Colours

If the field of vision is tested with a white and a coloured object of the same size against a black background, the coloured object will give a smaller field since it is a less strong stimulus and thus the field for colours is usually regarded as smaller than that for white. The consideration of this subject involves many extremely complex and difficult physiological questions, and in the present instance must be confined to the clinical and practical aspects. From the fact that a white object is seen as white in all parts of the peripheral field it follows that the fields for complementary colours, such as a complementary red and green or a complementary blue and yellow, are equal in extent. The charts usually given in text-books showing the colour field decreasing in size in the order yellow, blue, red, and lastly green in a comparatively small central area, refer to the results of observations made with one size of test-object, with colours which were not complementary and probably also varied in intensity of hue and in their white values. Unfortunately no standard of colours has yet been established, and it is because those in general use at the present time vary in regard to the points mentioned that the field for green is usually found to be smaller than that for red, and that for blue smaller than that for yellow, but if the intensity of the light, the saturation of the colour and especially the size of the object are adequate, these colours may be distinguished very nearly, if not quite, at the periphery. The extent of the field for each colour is also influenced by the nature and brightness of the colour to which the retina was exposed before the examination commenced (pre-exposure), and by the relation between the brightness of the background and that of the coloured object, the field being largest when the brightness of the object and that of the background are equal. With a black background, as is usual in clinical perimetry, the colour field will be relatively smaller if there is much white in the colour test used. It is of interest to note that if a coloured test-object, too small for the

colour to be accurately detected at the periphery, is presented there and moved towards the fixation point, the colour appears through several changes. Baird has shown that red first appears yellowish, then yellow, orange and orange red. Green passes from yellowish through greenish to green. Blue does not change in tone, but increases in saturation. These points are mentioned here because in pathological conditions a similar sequence of change may be observed. It is, however, possible to select colours which do not change in peripheral vision, the so-called "physiologically pure" colours.*

It is unnecessary as well as undesirable to overburden clinical perimetry with elaborations, which are of little, if any, practical advantage in diagnosis or treatment and, as far as concerns colour perimetry as a clinical method, the influence of a grey background and of pre-exposure may be neglected and we may regard the extent of the field for each colour as determined by the size of the visual angle subtended by the test-object against a black background. The positions of isopters for colour tests of different angles in the normal subject have not been fixed with any exactitude for several reasons, mainly because of the great variation in the results obtained from different individuals. Since the colour appears gradually as the test-object moves from the periphery to the fixation point, there is wide scope for difference of opinion as to when the object presents a definite hue, and the more difficult the test (*e.g.*, when the object is small) the greater are the variations met with. Ordinary differences in illumination or in the exact hue and intensity of the colours commonly supplied for the purpose are of little practical importance even when the objects are small. As the white value of the colours is usually too high, a relatively bright illumination tends to reduce the field more than a moderate or somewhat dull diffuse light, and should be avoided.

Using 20 mm. square objects at 330 mm., that is, with an angle of nearly 4° , the following figures were obtained by Berry :—

	Out.	Down.	In.	Up.
For a red and a complementary green	58°	44°	34°	28°
For a blue and a complementary yellow	72°	60°	44°	38°

With the perimeter and screen under ordinary clinical conditions in winter, the results shown in the Table on p. 12 were found by the author. The subject is further discussed in the Appendix, Note I.

These figures are serviceable as an approximate guide to the gradation of colour perception under the usual conditions present in the out-patient or consulting-room, and are not intended to indicate any scientific physiological values.

In practice, having regard to the hues which are commonly available, red is the most generally useful, blue is sometimes helpful in special cases, while green, though seldom required in itself, is useful as a contrast to red.

When the highly subjective and qualitative nature of the test is considered, it is

* See Appendix I., B, p. 289.

Visual Angle.	Object (mm.)	Distance (mm.)	Blue. (Degrees.)				Red. (Degrees.)				Green. (Degrees.)			
			Out.	Down.	In.	Up.	Out.	Down.	In.	Up.	Out.	Down.	In.	Up.
1-7'	1	2,000	4-4	3-5	4-4	3-2	2	1-8	0-2	1-3	1-6	1-4	1-3	1-3
3-4'	2	2,000	7-3	6-4	7-7	5-3	4	2-4	3-7	3-0	3-8	2-5	3-2	2-5
5-1'	3	2,000	11	11	13	8	6	4-5	6-5	4-2	5	3-0	4-6	3-0
8-6'	5	2,000	17	14	18	12	8	5	8	6	7	4-6	7	5-0
17-2	10	2,000	27	22	25	20	13	9	13	8	9	7-6	9-7	6-6
34-4'	20	2,000	33	28	32	27	20	13	18	13	15	10	14	9-6
1° 9'	40	2,000	—	—	—	—	32	20	20	19	22	16	17	14
10-4'	1	330	38	16	23	15	13	7	11	8	6	4	6	4
20-8'	2	330	50	20	26	17	22	11	14	11	13	7	10	6
31-2'	3	330	74	29	30	24	42	13	17	15	17	18	12	9
52-0'	5	330	80	33	35	30	53	21	23	22	28	12	14	12
1° 44'	10	330	87	47	43	37	79	35	29	31	49	16	21	18
3° 28'	20	330	89	55	46	41	85	46	36	37	65	21	22	21
6° 56'	40	330	92	58	48	43	87	52	41	42	70	30	29	29

evident that normal standards for peripheral colour vision can only be approximately indicated by the perimeter as ordinarily available. This, however, does not diminish the usefulness of colour perimetry as a valuable clinical method when used with judgment and discrimination in suitable cases.

It is necessary to remember that a test with colour as compared with white is a test with a stimulus, the strength of which has been reduced by diminishing its brightness but not its area. No special individual significance is to be attached to each colour and the field is never altered for one colour without being altered for all the others and for white.

The Blind Spot

The projection in the field of the head of the optic nerve forms a physiological blind area or negative scotoma, the blind spot or *area cæca*, which is oval in shape, with its long axis vertical. Under normal conditions insignificant variations occur in its position and size. In width it measures approximately 5.5° and in height 7.5° . Its centre lies about 15.5° to the lateral side of the fixation point and 1.5° or slightly more below the horizontal meridian, so that over two-thirds of its vertical diameter lie below the horizontal meridian. As this relationship is altered if the head is slightly tilted to one side, slight variations are easily produced and are of no importance. The distance of the blind spot from the fixation point is increased in hypermetropia and diminished in myopia in accordance with the difference in the length of the eye.

The dimensions given refer to the area of absolute blindness. Surrounding this there is a narrow amblyopic zone of about 1° in width in which modified vision is present both for white and colour. The degree of amblyopia is such that $\frac{1}{2000}$ or $\frac{2}{2000}$ white is not seen, so that the blind spot measured with such visual angles is about 2° more in total width than when mapped out by larger angles. With objects larger than $\frac{2}{2000}$ the

amblyopic zone becomes too narrow to mark out easily and its width is slightly greater for the smaller test-object.* Haycraft (158) found a much wider zone of relative amblyopia. The significance of this zone in clinical work will be referred to later (p. 67). Graphically expressed, the margin of the blind spot is steeply sloping at first, and later perpendicular, resembling a well with a crater-like mouth (Fig. 2). The area of absolute blindness corresponds not to the head of the nerve, but to the area in which no retinal receptive elements are present, an area usually slightly larger than the nerve itself. The presence of the amblyopic zone is at least partly anatomically explained by the gradual rather than

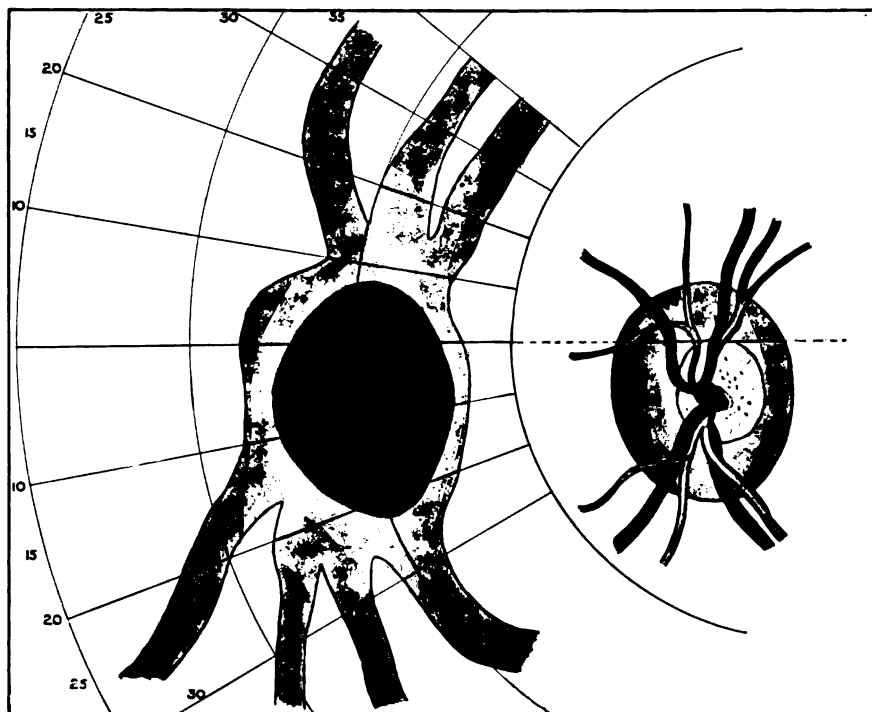


FIG. 6.—THE NORMAL BLIND SPOT (LEFT)

The drawing of the disc was made subsequently, and is turned so as to correspond with its projection. The amblyopic area ($\frac{30}{100}$) is about $30'$ wide on the nasal side, and 1° on the temporal. The projections of the vessels correspond closely with the true positions, but one vein and the smaller arteries were not detected. Test-objects, $\frac{2}{300}$ and $\frac{1}{2000}$.

abrupt termination of the retinal outer layers towards the nerve. At the upper and lower ends of the blind spot narrow curved prolongations of the amblyopic zone are found which represent the projections of the larger retinal vessels near the optic disc, and which with care may be traced some little way over the field even as far as 30° or more according to the observations of Young and Evans. Owing to the narrowness of the area covered by the vessel such scotomata are somewhat elusive, as even slight unsteadiness of fixation causes the test-object to "go out and in." Vessel scotomata are broader where they are joined to the blind spot and become narrower as they leave it. They follow the somewhat irregular course of the vessels rather than the more uniform curve of the

* Appendix II., p. 290.

nerve-fibres. They do not, except very superficially, resemble any pathological defect, and their relation to the blood-vessels can be easily checked by ophthalmoscopic observation.

These vessel scotomata are not often found unless specially sought for and do not form a usual feature of the $\frac{1}{2000}$ field. In order to demonstrate them the test-object must be moved very slowly and carefully. When tracing them towards the temporal side a 2 mm. object may be advantageous.

According to Evans, vessel scotomata are more easily demonstrated by the use of very small test-objects at short ranges, *e.g.*, $\frac{0.5}{1000}$, $\frac{0.25}{330}$. Evans used the Lloyd campimeter with objects from 1.5 to 0.125 mm. in diameter.*



FIG. 7.—SECTION THROUGH THE OPTIC PAPILLA, TO SHOW: (1) THE GRADUAL CESSATION OF THE OUTER RETINAL LAYERS; AND (2) THE COMPLETE TERMINATION OF THE RETINA SOME DISTANCE FROM THE NERVE.

These features of the blind spot show that it cannot be examined adequately unless at least two test-objects are used, at 2,000 mm. At a range of only 1,000 mm. the width of the amblyopic zone becomes too narrow to be satisfactory.

Between and including the fixation area and the *area cæca* is a horizontally oval portion of the field of great importance in pathological conditions, the *centrocaecal* area. These parts of the field correspond in the eye to the fovea, the optic disc, and the papillo-macular area respectively.

To the temporal side of the blind spot the field is slightly weaker than in other parts at the same distance from the fixation point. While $\frac{1}{2000}$ white is usually seen on the temporal horizontal meridian out to 26° , it is often easily lost in the strip between the 19° and 26° circles on the temporal side of the blind spot. The importance of this feature will be referred to in dealing with pathological conditions.

* "Angioscotometry" is the term given by Evans to the study of defects which are "angiogenetic in origin" and does not signify "merely a method of measuring retinal-vessel shadows." "Angioscotometry" is not dealt with in this work. A full account is given in Evans' Book, "An Introduction to Clinical Scotometry," by J. N. Evans. London. Humphrey Milford. Oxford University Press. 1938.

Conditions which Influence the Size of the Normal Field

The field is slightly larger in hypermetropia than in myopia, but the difference is not such as to have any practical significance for the clinical perimetrist. When using small visual angles, as with the screen test, uncorrected refractive errors may cause an apparent reduction in the field. The field may also be slightly diminished in extent by a small pupil or increased by a large one if the test-object is very small, owing to the difference in the amount of light admitted to the eye. With moderately large test-objects the size of the pupil makes no difference of consequence. Accommodation is said to increase the field by a few degrees, and as it is usually associated with contraction of the pupil the two conditions tend to counteract each other. While these circumstances should be borne in mind, they do not lead to results capable of being mistaken for evidence of pathological conditions.

The extent of the field is also affected by the illumination of the test-object, the contrast of object with background and the state of adaptation of the eye.* An object situated at the periphery of the field does not stimulate the retina as strongly as when it is placed near the visual axis because, in the former situation, less light is received from it. This is because of the obliquity of the incidence of the rays on the pupil which becomes relatively more and more slit-like the more peripherally the object is situated. The same factor applies to the surface of a flat test-object, when used with Bjerrum's screen, unless its face is turned towards the patient when it is peripherally placed. For our present purposes it is presumed that the object always receives average daylight illumination without direct sunlight, that the background is black and the eye light-adapted. Under ordinary circumstances such factors do not induce variations in the extent or intensity of the field of vision such as are likely to lead to errors in diagnosis. Quite a considerable reduction of illumination is required to produce a definite depression of the sensitivity of the normal field or to reduce its extent. The effect of dim light on the pathological field will be referred to later.

The term "normal field" is understood to refer to the whole area of the field including the central part, and not to the periphery only. In a *normal* field there can be no defect or depression of visual acuteness anywhere. The term "full" as applied to the field of vision should be avoided.

We are now in a position to affirm the characters—for clinical purposes—of the normal uniocular field.

1. The most peripheral or lowest isopter must lie in the normal average position ; in other words, the periphery must show normal, but not necessarily maximal, limits.
2. The internal or higher isopters must occupy normal average positions for the stimuli they represent, *i.e.*, the field must show a normal average slope.
3. The central peak of the field must attain an average normal height. This does not mean that the recognition of alphabetical or other symbols must attain to any arbitrary standard, but that the fixation area is to be regarded as

* For more detailed information see the papers by Walker, and by Feree, Rand, and Wentworth.

part of the field. The presence, however, of $\frac{6}{9}$ or $\frac{6}{6}$ vision with a normal field periphery does not necessarily indicate a normal condition of the central field, for normal central vision is sufficiently acute to undergo considerable impairment before an obvious reduction is demonstrable by a test such as Snellen's types. Where such impairment exists the patient is usually aware that his vision is not as good as previously, and the central isopters will be found altered in position, although the test-card may show excellent acuity. Similarly, expressions such as " $V = \frac{6}{24}$ field normal," are unsatisfactory if without explanation since the central field at least must be depressed unless there is some other reason for the impairment of vision.

4. Colour vision and the positions of isopters for colour tests, *i.e.*, the slope of the field for colour, should attain the average standard. Here a relatively wide allowance for physiological variation is necessary, and the capacity to recognise colours possessed by the individual must be taken into account, *e.g.*, congenital colour blindness may be present.

The practical criterion of the normal field will be considered in Chapter III.

CHAPTER II

PERIMETRIC INSTRUMENTS

The Perimeter

THE perimeter consists essentially of an arc of a circle supported on a convenient stand with a chin-rest or other appliance for keeping the eye in the same position throughout an examination and at a distance from the arc corresponding to its radius. The arc is placed with its concavity facing the patient, and is pivoted on the stand so that when rotated it describes a hemisphere into the hollow of which the patient looks, keeping his gaze fixed upon a mark—the fixation object—placed at the centre of rotation.

The line of vision is therefore a radius of the hemisphere, and at the same time the axis around which the arc is rotated. In whatever meridian the arc is placed all points upon it may be regarded as equidistant from the retina. The arc is marked in degrees from 0° at the fixation point to 90° or more at the free end.

A large number of different patterns of perimeter have been devised from time to time, varying more or less in detail though not in principle, and all claiming special advantages. No useful purpose would be served by attempting to enumerate or to examine the different examples in detail, but we may consider the more important structural features:—

1. The construction, extent, width, etc., of the arc.
2. The radius of the arc.
3. The desirability or otherwise of mechanical appliances for moving the test-object and for recording the field on a chart.
4. The stand.

The arc should have as a basis a strong but light piece of metal bent on its edge to the curve required. A strip of thin metal or wood is attached along the concave side of the supporting bar so that the completed structure has a T section with the vertical limb of the T on the convex side, or, better, two such bars may be used, giving the arc a half H section \sqcup ; either method of construction ensures strength, lightness, and permanency of curvature. Arcs made of yielding material, such as vulcanite, or made without a properly constructed rigid basis, are apt, after some time, to lose their original curve. The most suitable length of arc depends largely on the object in view. If it is desired to have a portable perimeter of minimum size and weight, an arc extending to 80° only may be made to suffice, pivoted on one end. For most purposes an arc of about 120° is more convenient, pivoted at 30° from one end so as to make a short and a long limb. This arrangement enables both sides of the fixation point to be tested up to the 30° circle in any one meridian without moving the arc, while by altering the position of the fixation object the available length of the arc may be increased.

The same object is sought in many patterns by the insertion of a circular plate about 20 cm. in diameter, often marked in degrees, at the centre of rotation, so that the central area of the field may be explored in every direction independently of the position of the arc. Such contrivances are, of course, merely methods of increasing

the extent of the arc, and are unnecessary if the arc is wide enough originally. If size and weight need not be considered the semicircular arc of 180 to 200°, pivoted on its centre, is the most suitable; it balances well and needs little adjusting, as a complete meridian can be examined in each position. The width of the arc should be not less than 75 mm.,* as it is desirable that the background should present as large a uniform surface as possible, and it should be faced with black woollen cloth without pattern, or painted with a really matt black surface. This point is of great importance, as other surfaces reflect light, and while quite suitable for rough examination with large objects, are

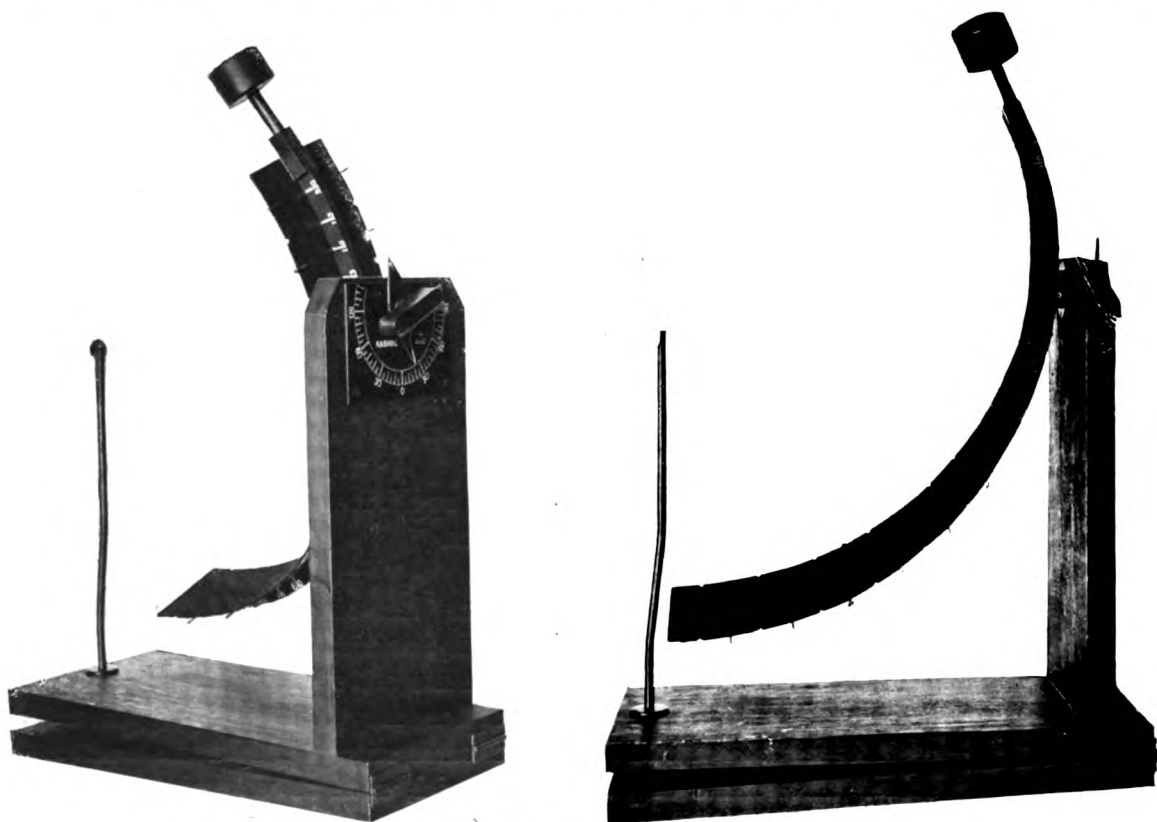


FIG. 8.—AUTHOR'S SMALL PERIMETER.

unreliable for fine work with small tests. The arc should be marked in degrees in such a way that the position of the test-object can be read off from in front. This is easily done when the arc is of half H section by placing the scale of degrees on the outer side of each rib. If the arc has a single supporting rib the scale is on each side of the rib where it is invisible from in front, but the position of the test-object may be shown by marking the tens of degrees with little knobs and the intervening fives with notches, while the 30, 60 and 90° points may be marked with slightly larger knobs. Such an arrangement does not disturb the regularity of the background, and provides the infor-

* Roenne (336) prefers a narrow arc used with Bjerrum's screen as a background. The wide arc is more generally useful, and should be placed in front of the screen when the latter is available.

mation required. A ratchet to fix the arc in meridians at, say, 30° intervals, facilitates accuracy and rapidity of working and is an advantage.

An important matter is the radius of the arc. Here again questions of expediency arise. The size and cumbersomeness of the perimeter increase rapidly with the radius of the arc, and if portability is desired, limitation becomes essential. In general, the radius varies from 250 mm. for small instruments to 300, 330 or 333 mm. for larger ones. Portable instruments, such as hand perimeters, are conveniently made with the smaller radius,

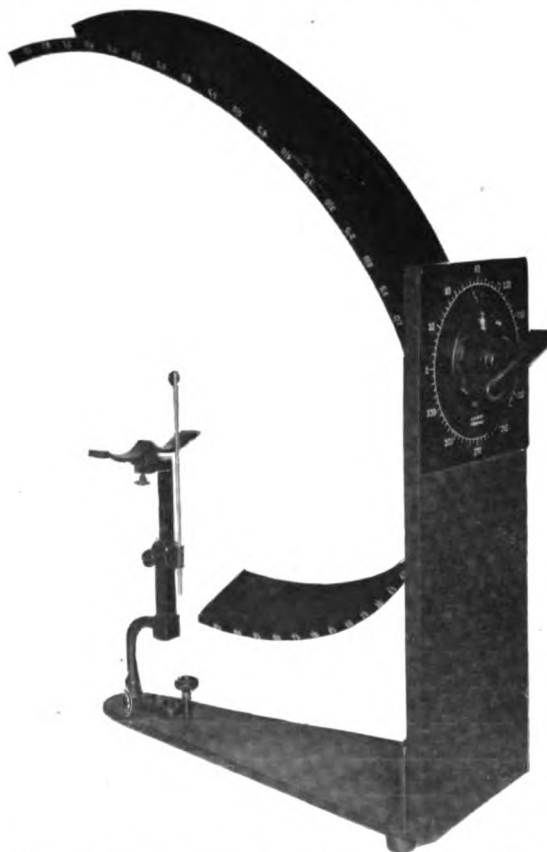


FIG. 9.—AUTHOR'S 330-MM. PERIMETER WITH HALF H SECTION \sqcup .
(Baird, Edinburgh).

while the larger is suitable for instruments which do not require to be much moved about.

Perimeters with a radius of a metre have been constructed (Elliot, Hudson, Mackay), and are suitable for more or less fixed permanent positions. They are inconvenient in clinical work and offer no advantage of value.

It has been suggested that the short radius perimeter imposes too great and continuous a strain upon accommodation, especially in presbyopes. There is no evidence that this occurs to such an extent as to hamper the examination or to give rise to misleading results. The field of the atropinised eye is not reduced in size, but rather increased owing to the greater admission of light.

The fixation object should consist of a disc attached at the centre of rotation of the

c 2

arc. The size depends upon the requirements of each case. A white disc with a black centre assists steadiness of fixation and helps to avoid fatigue. The disc should not glitter or dazzle, and for general purposes it may be made of matt white card or paper, about 3 mm. in diameter. At the centre of rotation of the arc a small hole, about 0.5 mm. in diameter, is bored, and the disc may be attached by passing an ordinary black pin, cut to a suitable length, through its centre. In this way the size and shade of the fixation object may be altered at will. In the case of a small perimeter it is convenient to have a similar hole at or near the extremity of the short limb of the arc, so that the fixation object may be placed there if desired. This has the effect of extending the arc to 100° or more, though it may measure only 80° from the centre of rotation, according to the distance along the short limb at which the fixation object is placed. It is undesirable to have a permanently marked and unalterable fixation object. Sometimes a small mirror is used. This may assist fixation in some cases, but is unsuitable for accurate work close to the fixation point.

We now come to one of the most important points in perimeter construction: Should the perimeter be "mechanical" or not? The mechanically actuated "carrier" holding the test-object and moving along the arc, and the automatic recorder for charting the field in their various forms are very attractive, and, theoretically, seem well adapted to the work. Opinions vary as to their advantages, and the writer is one of those who hold that such appliances are undesirable, and for the following reasons:—

1. The movements of the test-object are too limited. It is impossible to make the object appear at any part of the field or disappear at will, or to shake it or move it irregularly or suddenly change its colour, or to bring a small test-object close up to or past the fixation point. Accurate work close to the fixation point cannot be done with the usual form of carrier.
2. Too much time is occupied as the carrier moves relatively slowly, and rapid interchange of test-objects of various sizes and colours is impossible.
3. The carrier attracts the attention of the patient by reflecting light, and because it is not absolutely noiseless.
4. The surface of the arc becomes rubbed and tends to glisten. The carrier is not suitable for a cloth-covered arc or one with edge-markings.
5. The test-objects are easily soiled and troublesome to replace. In some carriers they are liable to be partly overshadowed by the edge of the hole in which they are exposed.
6. The apparatus is complicated, liable to get out of order, and adds greatly to the expense of the instrument.

The limitation of movement referred to under (1) is by itself sufficient to destroy the value of the mechanical perimeter as it is essential for the examiner to be able easily and quickly to check the patient's responses.

The first three points make it difficult both to avoid suggestion and to introduce it at will when desirable. The mechanical perimeter is, therefore, unsuitable for the examination of suggestible individuals.

It is difficult to think of any advantages offered by the carrier, and it is noteworthy that experienced perimetrists are practically unanimous in condemning it,* while the most important perimetric work has been done with the simplest non-mechanical instruments (*vide* Roenne, Walker, and others). Although it is true that the best modern perimeters are constructed so as to overcome some of the objections mentioned, yet no mechanism has been devised which affords the same facilities for ease, rapidity and accuracy of working as are provided by the manual use of several double-faced test-objects mounted on suitable holders.

The question of automatic recording apparatus is closely bound up with that of the mechanical carrier though somewhat simpler. The system by which a needle-point moves in unison with the carrier, enabling the chart to be pricked out, has all the defects mentioned above. A better plan is to attach the chart to the back of the perimeter so that it rotates with the arc, as in Priestley Smith's model, and can be marked by hand with a pencil. If the back of the perimeter is provided with a large revolving plate and a large chart is used, this arrangement has some advantages, and the circular scale of degrees at the back of the perimeter may be dispensed with, its place being taken by the chart. Limitations are, however, introduced, and the freedom of action of the perimetrist is somewhat curtailed, so that the advantages turn out in practice to be more than counterbalanced. In this matter, as in all perimetry, good results depend upon the worker rather than on his tools.

The stand should be light and preferably made of wood, and arranged so that the whole instrument can be tilted backwards in varying degrees. The upright upon which the arc is pivoted should be as broad as is compatible with portability and convenience, and it should extend several inches at least above the level of the axis of the arc. In this way a large background is provided which helps to obviate distracting influences, and enables the worker to collect his test-objects, charts, etc., in a position where they cannot be seen by the patient. It also provides ample room for a large circular scale of degrees to indicate the meridian in which the arc is standing. The slender iron pillar so commonly found—often brightly enamelled—seems to have been designed for appearance only.

A simple adjustable chin-rest, with a separate fixed point in the form of a button or knob which touches the lower eyelid so that the visual axis may be brought truly to the axis of rotation of the arc, completes the essentials of a soundly constructed perimeter.

The perimeter figured (Fig. 8) embodies the points mentioned, though capable of improvement, and is a modification of Priestley Smith's model. The radius is 250 mm. ; the chin-rest is omitted. Fig. 9 shows a 330 mm. model with semicircular arc.

As a comment on perimeter construction in general, it seems significant that students of perimetry, such as Roenne and Walker, should advocate the simple non-mechanical short radius type of instrument

The perimeter should stand upon a firm table, preferably with a black cloth surface, which can be raised or lowered as required.

Up to the present most perimeters have been constructed for use by daylight.

* *E.g.*, "Schnurperimeter sind auszurotten" (Roenne 345).

Satisfactory results may be obtained by using two lamps, one at each side of and slightly behind the patient, arranged so as to illuminate the arc. In the future it is probable that a definite artificial illumination provided by a lamp attached to the perimeter will be more frequently utilised (Fig. 11).

Special Perimeters

Many different forms of perimeter have been designed for special purposes. Amongst these we may notice the hand perimeter, a small compact portable form useful for examining fairly gross lesions in patients who are ill or bedridden, and meant to be held

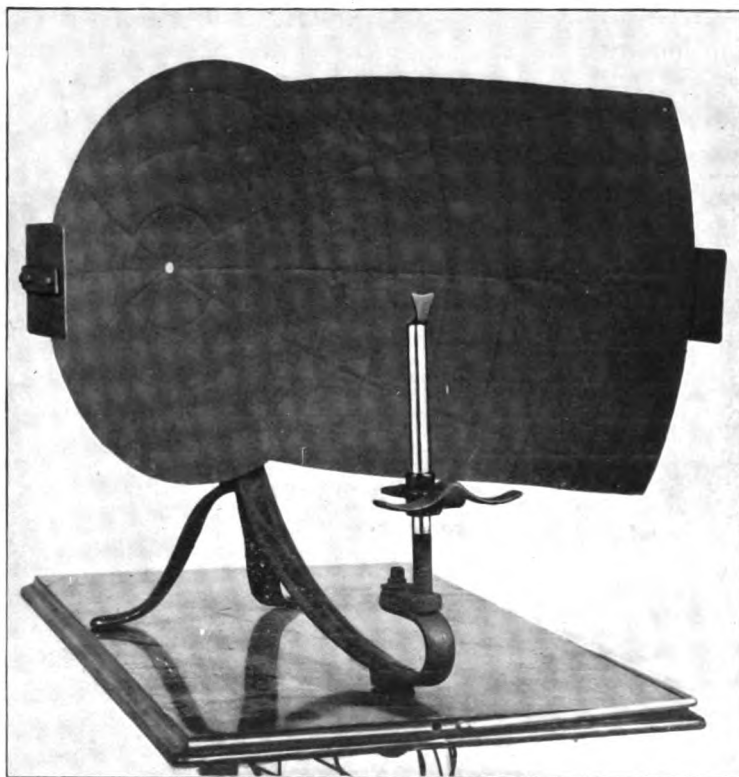


FIG. 10.—WALKER'S PERIMETER.

in the hand of the observer or patient. Perimeters with special stands for use with patients in bed have been designed, and also folding portable forms (Wilbrand, Holth, and others).

The development of neurological perimetry may be expected to largely increase the use of instruments adapted for the examination of patients in a reclining position in bed or in an armchair.

For general work an outstanding design is that produced by Walker (448). The instrument has no mechanical accessories. The radius is 28.6 cm., and a sheet of thin brass, curved to this radius, forming a considerable segment of a hollow sphere, is attached to the arc, so that not only one meridian, but a large area of the field may be

examined during one position of the arc, and also the test-object may be moved in any direction with great freedom. For hospital use this perimeter is attached to an overhead suspension apparatus in a special perimetry room, enabling it to be properly adjusted to a patient in any position whether seated or reclining.

The elaborate refinements of the Feree-Rand instrument have not yet been proved to be of special value in clinical work, while their utilisation entails the serious disadvantage of prolonging the time required for examination.



FIG. 11.—SELF-ILLUMINATED PERIMETER.
(Hamblin, London).

A recent perimeter, very scientifically and elaborately constructed but unfortunately very expensive, is that of Goldmann* (Berne). Its clinical advantages have yet to be determined.

Self-lit or electric perimeters have been advocated from time to time, but have not yet come into general use; they are apt to be elaborate and costly out of proportion to their clinical value. In such instruments the fixation object consists of a small spot electrically transilluminated, and the test-object is provided by a carrier containing a small lamp with coloured glasses and diaphragms of different sizes. In more recent

* Goldmann, H. "Périmètre sphérique à projection avec enregistrement automatique." Clinique ophtalmologique de l'Université de Berne, 1943.

models a beam of light, white or coloured, is directed on to the arc from a specially constructed lamp. As the instrument is to be used in the dark, mechanical recording and the carrier, with their limitations and inconveniences, are necessary, while it is impossible to observe, much less control, fixation. Electrically illuminated test-objects attached to the end of a rod for dark-room screen work have also been designed. The subject will be further referred to under dark-room perimetry.

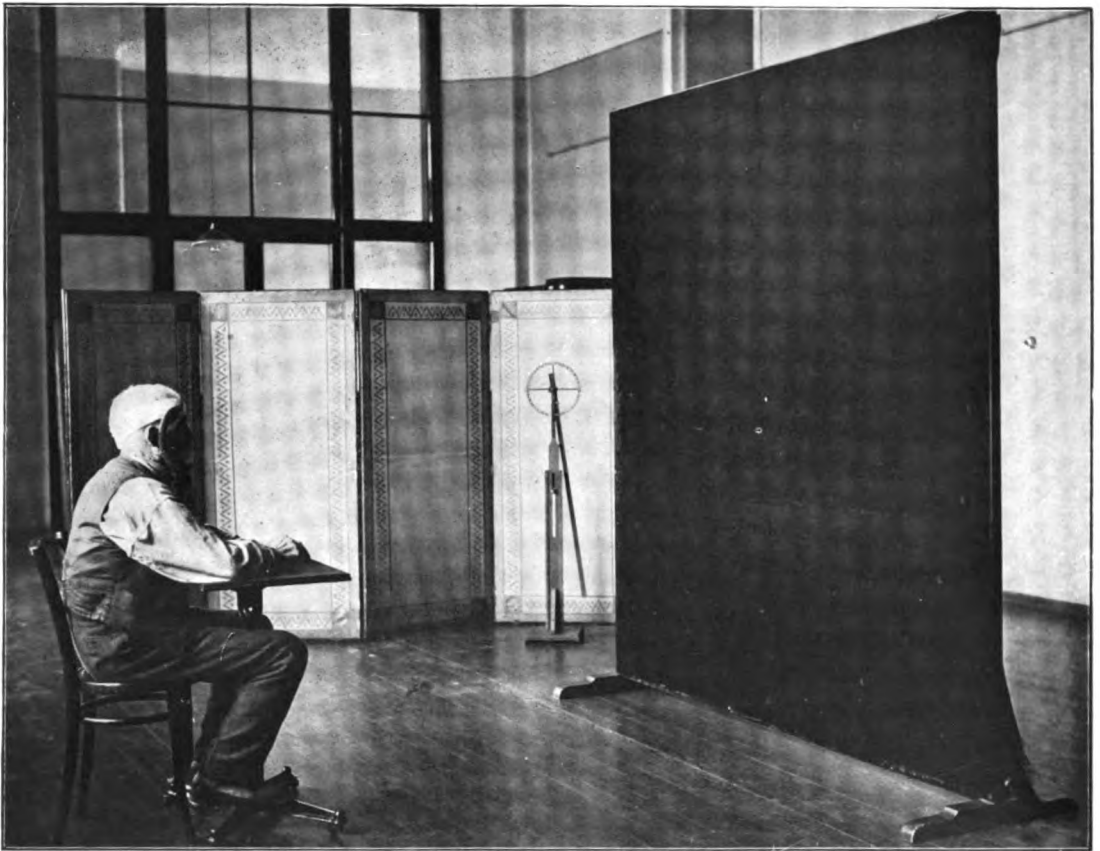


FIG. 12.—SCREEN EXAMINATION. CHIN-REST FROM CORNEAL MICROSCOPE.

The Screen, or Campimeter

The screen consists essentially of a square of black velvet or other suitable dark coloured material, without obvious pattern, and as free as possible from seams or markings, stretched upon a fixed frame light enough to be easily moved. The best size is 2 m. square. Roenne gives the dimensions as 2 m. high by $2\frac{1}{2}$ m. wide. Smaller screens may be used, but the larger size should always be preferred as it is incomparably superior as regards ease and accuracy of working. The fixation object consists of a disc of matt white paper or cardboard attached by a black-headed pin to the centre of the screen, which should be reinforced at this point by a small piece of felt stitched on at the back. This holds the pin firmly and prevents the development of a hole at the centre. The fixation object is usually about 5 mm. in diameter, but may be of any size suitable

to the case under examination ; it should not glitter or shine and it is well to have several discs of different sizes available, from 3 to 4 mm. upwards.

A small mark, invisible except by direct fixation, may be placed on each side at about the centre of the blind spot at 2 metres.

It is convenient to mark the edge of the screen at every 10° by a stitch of coloured wool, using different colours to indicate the chief meridians. The surface of the screen may be marked by a dark blue chalk pencil in concentric circles at 5° intervals corresponding to a tangent scale of degrees at 2 m. since the screen is to be used mostly at this distance. The vertical and horizontal meridians may also be shown. Such markings are almost invisible to the patient and facilitate charting of the fields. Some perimetrists (Roenne) mark the screen in concentric circles 10 cm. apart with the fixation object as centre. The value of these circles in degrees is then ascertained for each distance of the screen from the patient from specially prepared tables.* The fixation object must always be placed at the centre, and if the circles correspond to a tangent scale at a certain distance, they will only be of use at that distance.

A simple and convenient plan is to use an ungraduated screen and make the readings from a tangent scale appropriate to the distance adopted. In this way a screen of any size may be used at any distance with the fixation object attached anywhere on its surface, provided only that the requisite tangent scales, which are easily found from the tables † given by Maddox, are available. The only drawback to this method is the slightly longer time consumed in transferring the findings to a chart, but this can easily be done by a lay assistant (secretary or nurse).

A liberal supply of small black-headed pins should be at hand, and may be kept in a pincushion attached to the screen. The shafts of the pins should be enamelled or made of metal which does not corrode easily, as rough rusty pins are difficult to work with and damage the screen. When not in use the pins should not be left in the screen. Two or three varieties of pin with different sizes of head (which should, if possible, be matt and not glitter) are convenient for demarcating different isopters.

Walker, who made most elaborate studies of the mathematical considerations underlying perimeter and screen construction, designed a special combination screen consisting of two screens sewn together in such a way that three separate surfaces can be presented. These are marked for use at 1,500, 2,000 and 2,500 mm., so that no pins are required, and the screen is constructed with a curve, so that the usual error due to a flat surface is reduced to a minimum. Another form of campimeter with curved surface is the umbrella, which, though previously not unfamiliar to perimetrists, was also elaborated by Walker. An umbrella of 1,000 mm. radius provides a field of 45° , or more if eccentric fixation be used. Walker marked his umbrella for use at distances varying from 500 to 2,500 mm., and calculated the errors due to curvature and method of marking.

For exact work, such as is required when comparisons are to be made at a later date, a chin-rest is essential for use with the screen. It should be adjustable as regards the chin and forehead and the height from the ground, and must be comfortable. The

* Appendix, Table II., p. 301.

† Appendix, Table I., p. 301.

chin-rest used with the corneal microscope or slitlamp attached to an adjustable table is very convenient.

A great number of different designs of campimeter have been brought forward at different times. Some of these may be mentioned here.

The screen may be marked on the back and small steel discs coated with celluloid and controlled from behind the screen by a magnet are used as test-objects. By this method any confusion due to the object carrier is eliminated. This advantage, however, is only of value in connection with objects less than 3 mm. in diameter and other limitations are introduced.

Revolving scotometers have been designed by Priestley Smith, Elliot, and others. Here the test-object is fixed to the screen which revolves around the fixation object. In this way the circular traversing of the field by the test-object is facilitated and the patient is not distracted by the presence of a holder. This type of instrument, nevertheless, does not present any substantial advantages over the simple screen, as the limitation of the movement of the test-object hampers the freedom of the observer and is certain to influence the results. The ophthalmologist who possesses and can use a screen and a set of graduated test-objects will not find that anything is to be gained from the mechanical scotometer.

A miniature screen, such as a piece of matt-surfaced black cardboard or black cloth upon a stretcher about half a metre in diameter, is often handy for rough and rapid preliminary work.

Apparatus for perimetry or scotometry when fixation is bad, *e.g.*, when the scotoma is central and so dense that a fixation object of the usual size cannot be seen, is usually constructed with the object of making use of the sound eye. Thus the method of Schlösser depends on the use of a coloured glass (red or green) before the fixing eye, the field of the other eye being taken with a test-object complementary in colour. In Haitz's method fusion of two charts, one visible to each eye, is obtained by a stereoscope in the usual way, and the central field of the affected eye can be examined while fixation is governed by the sound eye. An excellent but expensive instrument of this type is the stereo-campimeter of Lloyd. Walker devised a "macular selector" and other instruments with the same object. Such appliances require very careful adjustment and great intelligence on the part of the patient if inaccuracies due to heterophoria are to be avoided, while they do not help if both eyes are defective. For ordinary clinical purposes, even when vision is well below $\frac{6}{60}$, satisfactory results will usually be obtained if the fixation object be made sufficiently large and distinct. Even when the fixation object is not well seen most patients of average intelligence can fix well and fairly steadily. This is partly because scotomata are not often absolute over the whole macular area and partly because the patient's sense of direction, in the case of an eye that has previously seen well, is very accurate. A patient with a hemianopic scotoma will say that when he looks "at" an object he sees only "half of it," but when he looks to one side he sees "the whole of it," and one with a central scotoma knows that he sees the fixation object better when he is not looking "at it." Thus, by getting the patient to find out where the fixation object is by using both eyes, or by paracentral vision, and then to look

“at” it, even if he does not see it best that way, quite satisfactory fixation can be obtained in most cases of central scotoma. In large and dense scotomata the patient may feel for the pin in the centre of the fixation object, and then fix upon that point. Marx advised a white ring with a black centre, and found that patients with central scotoma could fix the centre of such a ring to within 1 to 2°, an error which will rarely be of importance in regard to the field periphery. As central scotomata are examined at a much longer radius on the screen, with which fixation under adverse circumstances is, as a rule, more accurate than with the perimeter, errors due to unsteadiness are relatively diminished.

Test-Objects

1. *Shape*.—The object should be circular with two plane surfaces.
2. *Size*.—Each object should be of known area, and a set of objects should provide a range of stimuli suited to the various degrees of visual acuity present in the field. Sizes vary from 1 to 60 or 70 mm. in diameter.

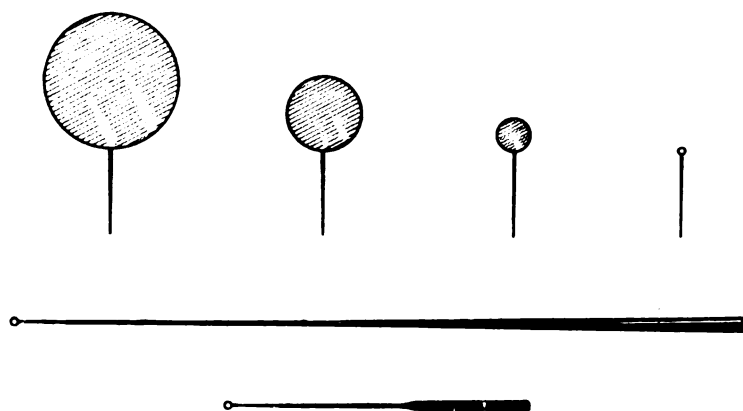


FIG. 13.—TEST-OBJECTS AND HOLDERS.

3. *Colour*.—White, red, blue and green tests are employed. White and red are the most useful colours. Blue and green are rarely required. The objects may be arranged so as to have a different colour on each surface, *e.g.*, white and blue, and red and green. Red-blue tests are occasionally useful. Such objects may be silently and almost instantaneously reversed, changing the visible surface.

The question as to exactly what tint should be selected is difficult, as it is necessary to utilise what can be readily obtained in the market. The hues should be as pure as possible, and should not contain too much white, though they should be sufficiently brilliant to be quite obvious to the average individual. If too much white is present, red is apt to be called yellow and green white, and it is difficult to evaluate the patient's responses, especially in hospital practice.

4. *Material*.—Matt-surfaced paper is in many ways the best material, especially for small objects. In some respects cloth is better, but it is unsuitable for objects below 10 mm. in diameter.

5. *Construction*.—The paper discs are mounted on thin wire stems about 2 inches in length which fit into the end of the holder.

6. The holder is a thin wand about a foot long for the perimeter and from 30 inches to 3 feet for the screen. In order to render it unobtrusive it should be as slender as possible, and to avoid undue vibration tapering is advisable, especially in the case of the long holder. Metal or wood or a combination may be used. The handle should be of wood and should have plane surfaces, and the thin end should be armed with a small piece of fine tubing into which the stem of the test-object is inserted so that the surface of the object is parallel to one of the faces of the handle. By this means the observer is enabled to know that the surface of the test-object is always correctly presented to the patient, a detail very easily overlooked. The holder and the wire stem of the test-object are painted matt black.

Properly made and properly managed holders do not distract the patient's attention from the test-object. In recent years projection apparatus has been introduced with a view to removing any source of distraction, but its disadvantages outweigh any possible gain.

Suitable test-objects, ranging from 1 mm. to 60 or 70 mm. in diameter, may be obtained from instrument makers, and it is well to have a supply of coloured and white discs for their renewal when soiled or damaged. Objects below 1 mm. in size are not to be recommended. When a visual angle less than $\frac{1}{330}$ (10.4') is required, it is better to use the screen or to test with colour. A set of white-blue and red-green tests of 1, 2, 3, 4, 5, 6, 7.5, 10, 15, 20, 30, 40, 60 and 70 mm. with 5, 10, 20, 40 and 70 in red-blue is ample for all requirements. For most purposes, 1, 2, 3, 5, 6, 10, 20, 30, 40 and 70 in white-blue and red-green will suffice; red-blue, if required, can be extemporised. In practice it will be found that while the 1 mm. white object is in constant use coloured objects less than 5 mm. are rarely required.

Test-objects must be kept clean, and if much used may need frequent renewal. Colour tests may be graduated either by keeping to one intensity of hue and varying the size of the object, or by varying the intensity of the hue while keeping the size of the object the same. While the latter method may be of value in special circumstances, the former plan is to be preferred on several grounds. With bold hues the patient can usually say fairly definitely whether he can name the colour or not, whereas, if the hues are pale, they are apt to be responded to in such a way as to leave the examiner considerably in doubt, unless he is very confident in the patient's intelligence and ability. Moreover, pale hues, if soiled, are unreliable.

Up to the present no standard colour tests have been generally adopted, but it is to be hoped that a solution of this difficulty will soon be obtained.

For special purposes the individual perimetrist may devise test-objects of various kinds, *e.g.*, those of Evans for "angioscotometry," which are made down to 0.25 mm. in diameter from thin silver wire.

Charts

Charts for recording the field should be as large as possible within practical limits.

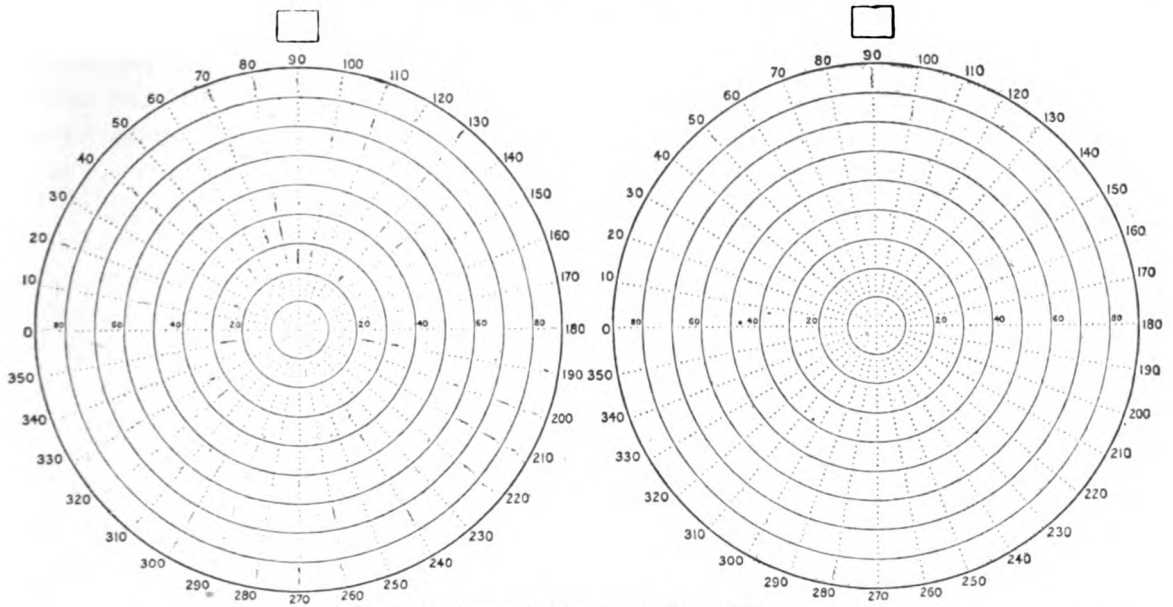


FIG. 14.—AUTHOR'S CHART FOR PERIMETER.

The paper should be smooth and thin so that tracings can easily be made. The printing should be done in red or terra-cotta ink, against which the inserted field will show up well. The radii should consist of lines of dots at definite distances apart and should be placed at intervals of 10° . In this way a point may be located to within half a degree. Separate charts on different scales may be used, a small scale for the periphery and a larger one for the central area, or two scales may be combined in the same chart.

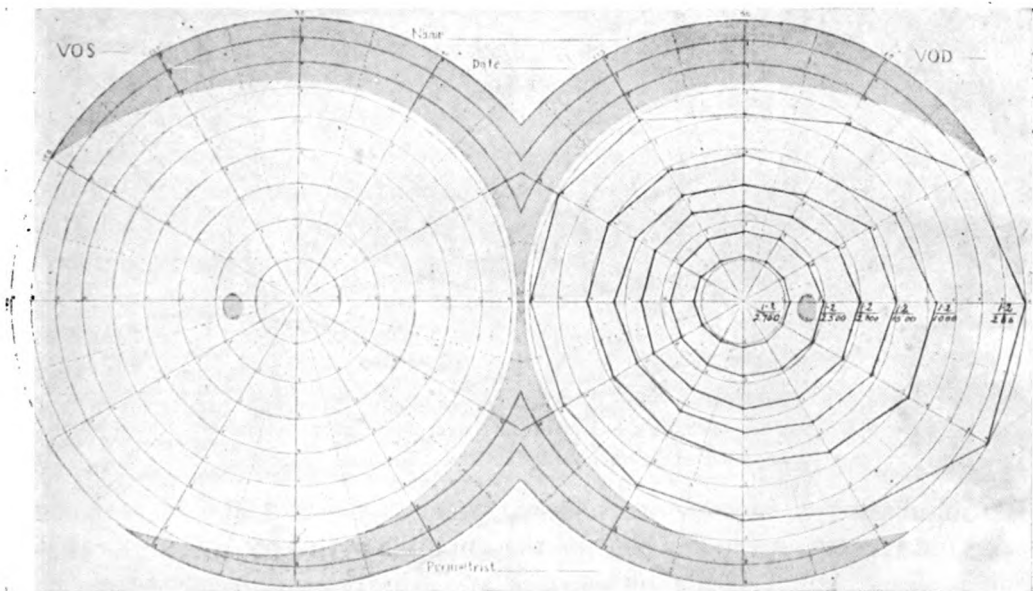


FIG. 15.—WALKER'S CHART.

Designed with larger scale centrally and smaller scale peripherally; showing also isopters as found by Walker.

If accuracy in the central area is desired, a scotoma chart on a specially large scale is necessary (Fig. 16). Each single chart should consist of a complete circle, and neither the normal field nor the blind spot should be indicated. This renders the charts available for either eye and encourages the production of an independent and unbiased record, and also keeps the chart free of undesirable markings.

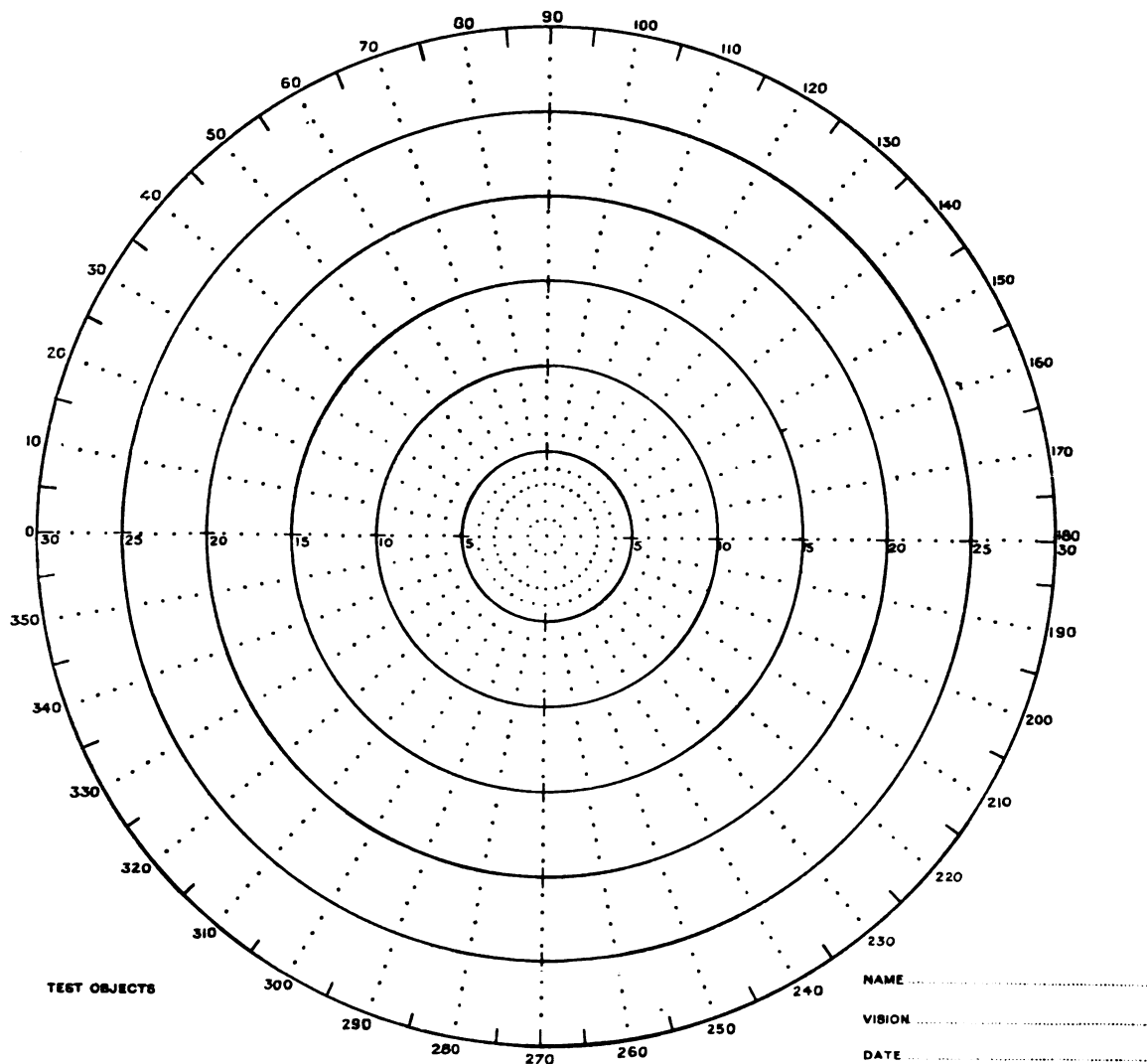


FIG. 16.—AUTHOR'S LARGE SCALE CHART FOR CENTRAL FIELD.
May be used for whole field if desired. Diameter 7 inches.

The numbering of the meridians should preferably correspond to that commonly employed for astigmatism and should be the same for both eyes. A uniform standard notation is desirable. The committee on Standardisation of Perimetry of the International Ophthalmological Congress, 1929, recommended that the notation should begin at the left end of the horizontal meridian and pass clockwise round the circle (Fig. 14).

As the field defect occupies a portion of the inside of an immense hollow hemisphere, it is not possible to design a chart upon which the shape, size and position of such defects can be truly represented. Charts have been designed (Roenne, 348) to compensate for this difficulty as far as is possible, but if it is remembered that the projection of the field on to the chart is purely arbitrary and that the defects marked out have not exactly the same shape as they have in the actual field of the patient, a frankly diagrammatic projection, such as that afforded by the commonly used Förster's chart, will be found satisfactory. This form (Figs. 14 and 16) has been used in the illustrations. From such diagrammatic representation the data may be transferred to other forms of chart if desired.

Since the field shown upon the chart is intended to represent the patient's subjective impressions the field of the right eye is placed on the right and that of the left eye on the left side of the chart with the temporal field of each eye to right and left respectively. This is known as the "physiological" representation of the field. Some perimetrists advocate the "anatomic" representation, placing the chart for the right field to the left of that for the left, as if the observer were looking at the patient's fields from in front and towards him. By this method a right-sided hemianopia is shown as a defect on the left side of the chart. All the fields shown in this book are drawn according to the physiological method.

For use with an unmarked screen a tangent scale is required.

Sinclair's rule consists of a strip of boxwood 1 m. long and about 25 mm. wide, marked on one side with tangents of degrees at 1 m., and on the other side with the same at 2 m. It is useful for measuring the distance between the patient and the screen and then for determining the position of the pins. For this purpose the angles may be obtained from the stitches placed round the screen, or from a protractor on a stand into which the rule fits. This is a handy and rapid method, as the rule remains in position while the chart is being marked.

Black gloves and a black gown and hood are accessories which are both desirable and useful, though perhaps more suited to research work than to the often unavoidably hurried examination of the clinical ophthalmologist. At the least, when using the screen, a white coat should not be worn, as this induces a disconcerting after-image. Where possible perimetry should be conducted in a room

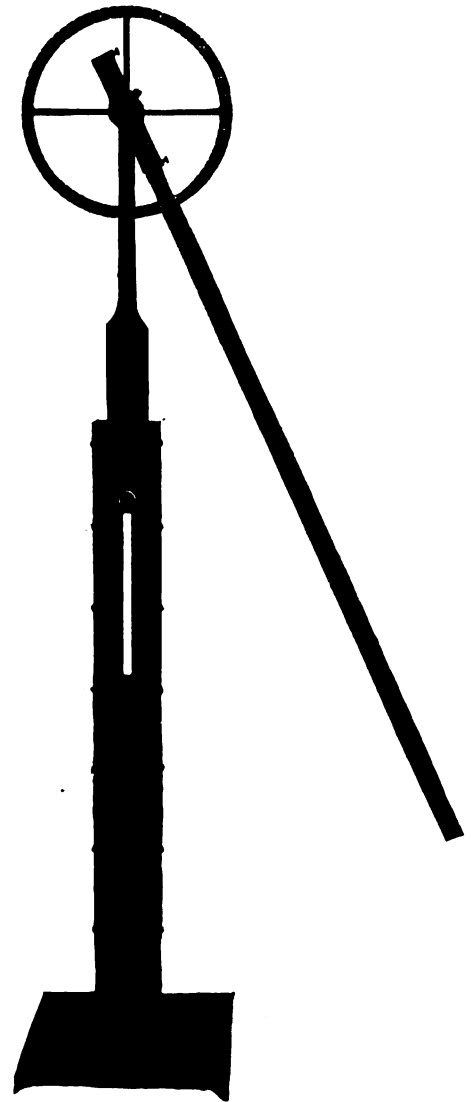


FIG. 17.—SINCLAIR'S RULE IN HOLDER FOR CHARTING FROM SCREEN.

set apart for the purpose and not in the dark room used for ophthalmoscopy and the slit-lamp. The walls should be dark, and, as far as possible, non-reflecting, and all bright or contrasting objects should be placed outside the patient's visual field.

The perimetry room brings us to the subject of illumination. What is required is an even illumination of the arc of the perimeter or the surface of the screen. The light should be steady and not too bright. For this purpose standard artificial illumination is the ideal method and may be obtained from a lamp of about seven foot-candle power attached to the perimeter. For colour fields a filter should be added.

If daylight is available the perimeter should be placed facing a window, preferably a very wide or a bow window, or, even better, between two windows, with a steady light free from direct sunshine, and for ordinary clinical work this will suffice. The windows should have suitable adjustable shades. Under these circumstances, however, it is obvious that the illumination of the test-objects will vary from day to day according to the weather and the hour, and also, on the same occasion, according to the position of the arc of the perimeter and of the object upon it. It is difficult to obtain uniform illumination of the surface of a large screen by the usual form of window. In any case, slight or moderate variations in illumination do not affect perimetric findings in such a way as to vitiate diagnosis, but it is always advisable to take precautions to reduce to a minimum the influence of this factor. This applies mainly to the estimation of progress by a comparison of findings obtained upon different occasions or to the search for faint traces of modified function by the use of very small test-objects. Even on winter afternoons good diagnostic work can be done if a single electric lamp is placed about a yard from each side of the patient's head.

While all such refinements in apparatus and method are undoubtedly of value, and perhaps especially in regard to research, their usefulness or, at the least, their necessity must be estimated with a due sense of proportion and perspective. It must be remembered that perimetry is not an exact science, but a subjective test, and that in any case, even with the most accurate apparatus and illumination, the cornea, pupil and lens of the patient introduce factors whose influence varies in different cases, and in the same case according to the position of the test-object on the arc of the perimeter. The general result is that, with the same size of test-object, the stimulus to the retina is, on the whole, less the more peripheral the position of the object. This is obvious in regard to the pupil which presents a more or less slit-like aperture to rays from a laterally situated source.

In reality, as pointed out by Roenne, the difficulties of perimetry, such as they are, are not physical, but subjective. The patient presents a complex of variable mental and physical factors which cannot be controlled and are difficult to estimate. To these must be added the personality of the examiner himself. Therefore, although strict precautions in regard to matters which can be regulated are wholly desirable, such factors should not be permitted to dominate the attitude of the perimetrist, who should never allow the excellence of his apparatus to govern his interpretation of the results obtained. Perimetry is not done by the perimeter but by the perimetrist, and simple tools properly used are much less productive of wrong conclusions than undue reliance on the dicta of an elaborate instrument.

CHAPTER III

METHODS OF EXAMINATION

BEFORE the field examination is commenced it is presumed that refractive errors have been estimated and that both distant and near central vision have been tested. The cornea and lens should be carefully searched for opacities, which sometimes, especially when small visual angles are being used, cause puzzling field defects.* The vitreous humour and the fundus, including the optic disc, the blood-vessels and especially the macular area, should be closely scrutinised. This examination is, naturally, not necessary in every detail in every case, and is better undertaken, if possible, some time before the field test, so that the patient may have an interval of rest.

Essentially there is only one method of perimetry. Each field is examined separately. The eye being "fixed" by suitable means, an object is moved about in various directions in the field at a uniform distance from the eye and the areas in which it is seen are demarcated from those in which it is invisible.

Many refinements of this basal method have been devised with, as we have seen, special appliances for each procedure. The most useful methods of field examination may be considered under the following headings :—

- (1) The confrontation or direct method.
- (2) Examination with the perimeter.
- (3) Examination by the screen or campimeter.
- (4) Special methods.

Whatever method or combination of methods is adopted, the principle of quantitative testing should be adhered to, and the examination should be planned to elicit the intensity of any defect which may be present, as well as its position and extent.

(1) **The Confrontation Method.**—The patient stands with his back to a window, having the eye not under examination covered by a suitable light bandage or by the palm of his own hand. The better eye should be tested first, as in this way the patient more readily grasps the nature of the procedure. The examiner stands about a yard in front of and facing the patient, who is instructed to look steadily at, or *fix*, the examiner's opposite eye. If the vision is too much impaired to enable the patient to see the examiner's eye he should look at the examiner's face, or in the direction of his voice. The examiner watches the patient carefully to see that fixation is steadily maintained. A test-object mounted on a holder is now introduced at the periphery of the patient's field in the horizontal meridian and moved across to the other end of the meridian keeping it always at about 18 inches from the patient's eye. The patient is instructed to indicate the point where it is first seen by him and also any subsequent points of disappearance and re-emergence. He should not be relied upon to volunteer statements, but should be constantly asked whether he sees or does not see the test-object in different parts of the field. This process is repeated in as many meridians as

* The consideration of field defects due to medial opacities is omitted, but the possibility of their presence should not be forgotten. Peripheral or irregular changes may be found often showing contradictory and inconsistent features.

may be considered necessary, not less than four. A white disc 20 to 60 mm. in diameter mounted on a suitable holder forms a useful test. If an object of this size cannot be seen the examiner's hand may be used or a sheet of white paper. Large coloured objects may also be employed. The test-object should be zigzagged slowly and moved in various directions as well as centripetally, or the fingers may be alternately opened and closed, thus enabling confirmatory questions to be asked. Circular movements are often valuable. If possible the examiner should have a dark wall behind him. An approximate estimation of the condition of the field is thus obtained and the examiner is at the same time able to observe whether fixation is steady. Useful information may be obtained in many cases, such as hemianopia, detachment of the retina, or glaucoma where the affected area is nearly or completely blind, and also often in cases of central or paracentral scotoma.

Suitable modifications of the confrontation method are often useful. Of great value is reduction of illumination. The examination may be carried out in the dark room with the light so arranged that the examiner's hand or test-object is only faintly illuminated. If the patient is not able to see the observer's eye he should look at his face or to his own front or in any direction the observer may indicate. Much useful information may often be obtained when vision is greatly reduced by simply moving a large white object about in the field while the patient maintains fixation as steadily as possible.

In suspected homonymous hemianopia the patient may be asked to look at the observer's face with both eyes open while the latter holds up both his hands, one in each temporal field, and asks whether one or two are seen and, if two, whether both are equally distinct.

The confrontation method is, however, admittedly crude, and for the most part only capable of roughly indicating the presence of comparatively gross lesions. In less obvious cases unsatisfactory results may easily be obtained. For this reason any elaboration of the confrontation method is undesirable as tending to elevate it from its position as a crude preliminary test for gross defects. As such it has a definite though limited sphere of usefulness in cases in which closer examination is unnecessary, and in pointing to the need for further investigation in others. When intelligently and carefully used almost as much and as valuable information can be obtained in some cases by this method as by more elaborate and delicate tests, but when too rapidly or roughly applied, especially by an inexperienced observer, it may be valueless.

The examination of children and of mentally affected persons may be carried out by modifications of the confrontation method. As a rule, it is best to confine the examination to a test of the binocular field for a gross defect such as hemianopia. While the child is looking in a certain direction an object may be introduced into the lateral field and the readiness with which its presence is appreciated compared on the two sides. Or an object which the child desires and can follow with its eyes, such as a toy or sweetmeat, may be held before it and then suddenly jerked into the lateral field while the behaviour of the child is noted. In the case of paralysed, unconscious, or otherwise mentally affected persons, a light may be suddenly flashed laterally upon the eyes in order to evoke reflex closure of the lids, which does not occur if the light is

in the blind part of the hemianopic field (Wilbrand and Saenger). In both children and adults external disturbing influences should be avoided and the results must be interpreted with caution.

(2) **Examination by the Perimeter.**—The confrontation method is, in practice, an almost purely qualitative test. With the use of the perimeter the quantitative examination of the field is commenced, comparable to the use of Snellen's types for central vision. In this case, however, it is not the ability to distinguish two points as separate that is tested, but the power of recognising the appearance and movement of objects apart from their form, which is the special function of the peripheral retina. At the same time, whatever diminishes the power of perceiving the clearness of a single object, diminishes also the power of perceiving two points as separate, so that quantitative perimetry may be regarded as the testing of the acuity of indirect vision. In the light-adapted eye, peripheral vision differs from central vision in degree rather than in character.

The quantitative element is introduced by the systematic use of a series of test-objects of graduated sizes, so as to determine for each part of the field as nearly as possible the minimal stimulus required to excite vision.

The patient must be comfortably and easily seated so as to avoid all fatigue or strain and to permit him to conserve his attention for the business in hand. Before him a black screen (Bjerrum's) should be placed. In front of this the perimeter stands on its adjustable table, so arranged that when the patient's chin is on the rest a line connecting his eye to the fixation object will be a radius of the hemisphere described by the arc. The eyes should be in the same horizontal plane so that the vertical and horizontal meridians of the projected field may coincide as nearly as possible with the vertical and horizontal positions of the perimetric arc. This precaution is of importance in the delimitation of hemianopic and quadrant defects and the nasal step of Roenne.

The lighting should come from behind and from the sides as already described. The eye not under examination should be covered by a light pad not projecting beyond the bridge of the nose, or, if much perimetry is being done, a special blinder such as that of Walker may be found advantageous.

The examiner should stand for the most part to the side of the perimeter corresponding to the eye not under examination, facing approximately in the same direction as the patient, and taking care not to obstruct the light falling on the test-object. In this way the right hand is used to hold the test-object when examining the right eye and the left hand for the left eye.

It is well to adopt a definite system in regard to procedure. Having explained to the patient the nature of the examination and shown him the test-object, the examiner makes a few preliminary trials in order to see that the patient is co-operating, and in order to give him a very short but necessary training. A fixation object of about 3 to 5 mm. in diameter should be chosen if the patient's vision is good; if not, a disc large enough to be fairly well seen may be used. Smaller fixation objects are not desirable for prolonged use as they tend to blend with the background, owing to the physiological

unsteadiness of fixation. For the same reason it is inadvisable to work with a small white test-object close up to the edge of a much larger white fixation object. A white test-object not larger than 3 mm. in diameter may be selected to begin with, and the first few trials should be discarded. It is well to begin with the eye which has better visual acuity, or, if there is no difference, with the right eye on the horizontal meridian, preferably at the temporal side, as this gives the examiner an opportunity of verifying both fixation and cooperation by noting the blind spot. The test-object should be placed near the blind spot and moved slowly across it in various directions until the patient realises that he has a blind spot. The discovery of an area in which the test-object disappears when he keeps his eye steady enlightens the patient as to the nature of the examination, shows him what is meant by fixation, and helps to secure his cooperation. If the blind spot cannot be demonstrated on the perimeter an attempt should be made with the screen, returning afterwards to the perimeter. In the rare cases in which difficulty still remains the patient must be regarded as unreliable. This preliminary test need only occupy a few minutes but is important.

The examination is commenced by placing the test-object before the arc at the temporal periphery and moving it inwards at a moderate and steady rate by a series of short, slow, zigzag movements across the line of progression. The best rate of movement along the arc is soon ascertained by experiment. If too fast the response will come late. This will be discovered by moving the object in the opposite direction over the same area, when a considerable discrepancy may be found. A rate can then be adopted which is suited to the patient's reaction time and gives results which show a minimum of dependence on the centripetal or centrifugal movement of the object. For this reason the first few trials are not recorded. When the perimetrist has estimated the mental ability of his subject the examination may proceed.

The object is carried up to within 20° of the fixation object in each meridian. Since it is easier and more accurate, as well as less trying for the patient, to examine the centre of the field by the screen, the test-object need not be brought closer to the fixation point than 20° . If no screen is available the test-object should be carried slowly up to the fixation point checking the results carefully. The patient is instructed to respond in monosyllables, and to indicate the appearance of the test-object by "now" or "yes," disappearance by "gone," reappearance by "back." It is not sufficient merely to ask the patient to state when the object comes into sight or disappears, he must be stimulated by the constant repetition of expressions such as "Do you see it yet?" or "Do you see it still?" More time and energy are saved than lost by adopting this precaution. In the writer's experience response by tapping is not so satisfactory.

Care should be taken not to let the test-object touch the arc as it soon becomes soiled and useless if this is permitted.

In this way the field is examined in a clockwise direction on meridians at intervals of 45° or, preferably, 30° .

Centrifugal movements are usually secondary and supplementary, especially with the small visual angles. In certain cases, as when examining functional field changes, centrifugal movements are frequently employed. When a defect is found, the exact

position of its boundary is best defined by moving the object to and fro across and at right angles to its margin until reasonable accuracy can be assured. As a rule it is better to move the object from the blind to the seeing area. Circular movements are very useful for defining boundaries which lie approximately radial in direction, such as the edges of a wedge-shaped or quadrant defect. The arc is rotated to and fro while the test-object is held before it at successive points. The necessity for such to and fro movements is, of course, greater when the edge of the defect under examination is sloping than when it is abrupt, and it is due to a factor which influences all perimetric examinations: the boundary under examination tends to be displaced in the direction of the movement of the test-object. It is, as it were, pushed onwards by the test-object, and this applies whether the object is moved from the blind to the seeing area or vice versa. In the former case the seeing area becomes diminished, in the latter increased.

The blind spot, for example, when mapped out on Bjerrum's screen, is usually found a little smaller by movements towards its centre than by movements in the opposite direction, and the highest degree of accuracy is obtained by a combination of both at the margin. A patient will not infrequently state that the object really appeared or disappeared some time before he said so, rendering a repetition of the test necessary. This tendency is physiological and concerned with matters such as the attention and alertness of the patient and his reaction time. It is evident that rapidity and facility in making variations in the movement of the test-object are better attained when the latter is held in the hand than when it is subjected to the limitations necessarily associated with mechanical perimeters or scotometers.

A test-object of the size suggested (3 mm.) affords a visual angle of about 0.5° , according to the radius of the perimeter, and is sufficiently large to give nearly the maximal normal field, while small enough to ensure the detection of defects of moderate intensity. If doubtful or blind areas are found, the stimulus may be increased by giving the test-object a more or less active oscillating movement. Areas in which the object is only seen during active movement may be noted as dim or doubtful for that visual angle.

The intensity of any defects found is then tested by means of larger objects up to 50 or 60 mm. in diameter if necessary, bearing in mind that the larger the object used the more care is required in fixing a definite limit, as the edge of the object may be seen while its centre is still in a blind area. The seeing field found previously is then tested in the same way with a 1 mm. object, giving an angle between $11'$ and $13'$, according to the radius, in order to elicit or exclude defects of lesser intensity. Other test-objects may be used according to the requirements of the case; the method described is a practical and satisfactory routine in the first instance.

Time will be saved and the patient's power of response conserved by concentrating attention on defective areas so as to bring out features of diagnostic importance while treating the remainder of the field more superficially, but still adequately. Thus, in a case of relative homonymous hemianopia, for example, in which the state of one side of the field requires the use of several test-objects for satisfactory examination, it is only necessary to demonstrate rapidly the integrity of the other side, its precise extent is rarely a matter of importance.

The Examination with Colours

Colour testing is carried out in a similar way with objects of graduated sizes. Although many of the results obtainable by colour tests are perhaps more easily and more accurately secured by the use of small visual angles with white, this method by no means supersedes the use of colours, which, in certain types of defect, give valuable information. The substitution of colour for white is an easy and convenient method of reducing the strength of the stimulus without reducing its area, which in fact is increased as the test-objects used are larger.

Coloured objects may be used in two ways : the recognition test and the visibility test. These two methods should not be confused.

The colour recognition test requires the patient to indicate when the test-object acquires or loses its true colour. Thus a red object moved towards a defect which has a sloping edge is first seen as red, then pink, then yellow and may finally disappear altogether if the defect is dense and the object not too large. When the object is moved away from the defect the order of the colour changes is reversed. The edge of the defect is marked at the point at which the red colour begins to fade or to appear definitely recognisable.

This test makes a relatively severe demand on the patient as it is easier for most persons to say whether a white test-object is visible or not than to state the moment when an object already visible and of apparently indeterminate colour acquires a definite hue. To obviate this difficulty Roenne adopts a " comparison " test and makes the criterion of the limit of the red field, for example, not the point where the patient thinks he can recognise the red colour, but the point where he can tell red from green. The patient should be shown the colours beforehand in large objects to make sure that he can recognise and name them, and the observer must satisfy himself that the colour is recognised by its hue and not by its shade. During the test it is better that the patient should not know what colour is being used, hence the advantage of having the objects differently coloured on the two sides, so that the colour may be instantaneously and silently changed. As with white, a few preliminary observations should be made, and the first few results discarded. The test most useful to commence with is a 10 mm. or 5 mm. red. The same zigzag movements are employed, and the point at which the colour is recognised, not that at which the object is seen, is noted. Different sizes or different colours may be used as the occasion indicates. The examination should be carried out as rapidly as possible, omitting unnecessary work on important parts of the field, as prolonged colour testing is apt to give rise to unsatisfactory results.

The colour visibility test uses the coloured object as a target which reflects less light than a white object of the same area, simply as a relatively dim object. The edge of a defect is marked at the place where the object becomes visible or invisible according as the object is moved from or towards the densest part of the depressed area. This test offers no difficulty to the patient.

The colour visibility test gives a much larger field for the same size of object than the colour recognition test. Thus the field for 2/2000 red by the former method is little

smaller than that for 2/2000 white while by the latter it extends to only a few degrees. (See p. 12.)

Both methods have advantages in certain types of impairment of visual response and in difficult cases both methods should be used and their results compared.

Colour testing is a part of quantitative perimetry, not a different kind of perimetry opposed to perimetry with white objects. It has the advantage that a delicate test is provided with a comparatively large object. It cannot be entirely replaced by the use of small visual angles with white since it differs from this mode of examination in one important respect. In colour testing a relatively weak stimulus is applied to a large area of the retina ; in the case of the small white test-object a relatively stronger stimulus is applied to a very minute retinal area. Colour testing is therefore more closely related to testing by reduced illumination.

Colour testing is chiefly useful for searching for defects of very slight intensity, for examining doubtful areas found by white tests, for studying the relation of the colour fields to those for white (see p. 62) and the quality of relative defects, and for making sure that an apparently normal field really attains the normal level. Its value should be estimated on the basis of the field changes as a whole, not on the results of the colour tests alone.

Recording

Recording is done by marking the field on a chart by hand, memorising several meridians at a time, or, better, by dictating the figures to an assistant who notes them down in sequence and at the same time sketches in the field. It will be found best to depict the field as it appears to the patient (see Chapter I). The name of the patient, vision with correction, and date should be included. The sizes of the objects used must not be omitted, and are best noted as already described by using the object as the numerator and its distance from the eye as the denominator of a fraction, a method which has the advantage of indicating all the factors concerned.

Perimeter examination should be regarded as mainly a method for determining the condition of the peripheral zone of the field. The central area and the blind spot may be quickly explored, but the patient's endurance should not be tried by spending too much time in working out intricate or difficult details in these parts as they are much more rapidly and efficiently dealt with by the screen.

(3) **Examination by Bjerrum's Screen.**—Following the examination by the perimeter, the central 30° of the field should be investigated with suitable visual angles. Small angles are obtained by increasing the distance of the test-object from the eye up to 4 m. or even more, if necessary. Roenne works at 2 m., which is undoubtedly the most satisfactory distance for the great majority of cases. In neurological work Walker found the most useful distances to be 1,000, 1,500, 2,000 and 2,750 mm. The greater the distance the larger is the projection of the defect, but beyond a certain point practical inconveniences arise.

The purpose of the screen test is not merely to diminish the stimulus, which can be done in other ways, but to increase the area covered by the projection of defects so that they may be more easily and accurately mapped out and analysed. It is only in

this way that the details of large central defects and the size and shape of small ones can be demonstrated. At the same time the screen test should never be regarded as replacing, but as supplementary to ordinary perimetry.

The 2-m. screen at a distance of 2 m. is greatly to be preferred, though much can be done with a smaller screen at 1 m. if space is restricted. For the detection of early or doubtful changes a working distance of less than 2 m. is of little value. The method is the same in either case, and, in the following description, it is assumed that a 2-m. screen at 2 m. is being used.

The patient is comfortably seated at the distance selected and the lighting is arranged as for perimetry. Care is taken to see that the head-rest is comfortable, though for rapid work in simple cases the rest is not essential. A fixation object adapted to the visual acuity is chosen. A dull grey disc about a centimetre in width, but larger and white if vision is poor, is suitable, and is attached to the centre of the screen by a pin with a dull black head about 3 mm. in diameter, so that it appears as a circle with a black centre. For fine work close to the fixation point the disc should be very small. If the centre of the screen is not quite on a level with the patient's eye no great harm ensues as the error introduced is mainly at the periphery of the screen and is very small at 2 m. This error may be compensated for by slightly tilting the screen forwards or backwards to bring its plane perpendicular to the visual axis.

If small objects are used, the patient's refraction should be corrected for the distance, and the appropriate lens, which should be circular and large, is conveniently attached to the brow by adhesive plaster, or may be placed in a frame so arranged as not to interfere with the visibility of the test-object.*

The examination should again be systematic, commencing with the right eye, if it is to be examined, and beginning with the blind spot. A few minutes should be spent in instructing the patient and showing him the test-object.

A white test-object of about 40 mm. is suitable. It is first held near the edge of the screen on the temporal side of the field slightly over 20° from the fixation object and about 5° below the horizontal meridian. Having ascertained that it is seen there, it is moved towards the fixation object, when it should disappear at about 18° and reappear at about 13° from the centre. At these points pins are rapidly inserted by the unemployed hand, and the observer makes sure that the test-object is not seen when placed between them. Then from this point as centre the blind spot is outlined by moving the test-object from its centre to its periphery. The edge of the 40-mm. test-object does not come into sight as a sharply-cut curve, but as a slightly diffuse white glow. The patient should be warned to expect this and should be asked to indicate the moment when this glow appears and not to wait until a large part of the object is clearly visible. The point where the glow becomes visible should be marked by a pin until the blind spot is demarcated by eight or more pins at approximately equal intervals. Should the position of the edge appear doubtful at any part a to and fro movement across the doubtful line should be employed until the exact boundary can be determined. It is essential to

* The use of a lens in this position slightly alters the visual angle subtended by the test-object and the extent of the field found (see Roenne, 336).

commence the examination of the central field on the screen by the location and demarcation of the blind spot. The establishment of the correct position of the blind spot indicates that the patient's fixation is accurate. Inaccuracy is almost invariably due to wandering fixation caused by inattention or involuntary following of the object with the eye. At 2,000 mm. the blind spot and its amblyopic zone can be so accurately mapped out that observations made by different individuals or at different times will usually be found to coincide within a small fraction of a degree.

If no defect has been found by the perimeter, the field is now mapped out with a 2-mm. or 1-mm. white test (visual angle 3·4' or 1·7').* The object should be moved at a rate less than 1 foot per second, with little excursions at right angles to its line of progress. With centripetal movements carried up to the fixation object in this way the boundaries of the field are pinned out rapidly at intervals of 45 or 30°. A few centrifugal movements are often useful at first in order that the patient may thoroughly understand the examination. If at any part a pathological change appears to be present, attention should be concentrated upon it, and tests made at intervals of 10° or even less. The test-object should be moved radially, circularly, and in any and every direction which may suggest itself to the examiner for the better elucidation of the condition found. Movements at right angles to the edge of the defect will be found most profitable. The object should be moved from the blind to the seeing areas as the patient's fixation is less likely to be deflected than if he is able to follow the object until it disappears. The object is also apt to come more sharply into sight. If reaction time is slow, or if there is doubt, it is advisable to decide boundaries by to and fro movements at right angles to the edge of the defective area. Since the screen is a flat surface the visual angle subtended by the test-object becomes progressively less as the latter is moved away from the centre owing to the increase of its distance from the eye, though within the limits of the ordinary Bjerrum's screen the alteration is not serious. The strength of the stimulus is also reduced by the alteration in the angle of reflection of the light from the object to the pupil and by the relatively slit-shaped aperture offered by the pupil. At 1 m., if the screen is 1 m. square with the fixation object in its centre, the test-object at the edge of the screen will be about 12 cm. further from the eye than if held near the fixation object and nearly 24 cm. in the case of the 2 m. screen. Beyond the limits of the 2 m. screen the error increases so rapidly that the use of a flat surface becomes undesirable. If it is desired to examine the peripheral field by the screen, the temporal and nasal parts may be investigated by arranging a separate fixation object and placing the screen towards the side of the patient at a suitable angle, a troublesome method and one which is rarely, if ever, required.

The test-object should therefore be brought a little forwards when working near the edge of the screen and its face should be turned slightly towards the patient. Care should be taken not to let it touch the screen surface so that it may not become soiled.

* The 2-mm. object forms a distinct and reliable test, and is, perhaps, preferable to the 1-mm. for those who have not much experience of this method. In practised hands the 2-mm., or an even smaller test, is quite dependable. The patient should be warned to pay no attention to the holder.

The normal field for a $\frac{1}{2000}$ test should extend to nearly 26° all round, thus occupying almost the whole surface of the screen. The amblyopic zone of the blind spot should be determined. The internal limit of the field for $\frac{1}{2000}$ or $\frac{2}{2000}$ round the blind spot is often of great importance. No examination of the blind spot is adequate unless more than one test-object has been used. A close search should then be made for defects near the fixation area and around the blind spot.* The object may be passed several times vertically across the area between these two points to exclude arcuate and centrocaecal defects, and the field immediately above and below the horizontal meridian on the nasal side should be examined to exclude Roenne's nasal step. In clinical work the examination with $\frac{1}{2000}$ white is one of the most important tests and is indispensable. To omit the $\frac{1}{2000}$ test is comparable to examining central vision with a chart which has no line below 6/12.

Finally, colour tests may be used with the screen. They are valuable in the final search for defects whose intensity is so low that they are not disclosed by a white test as small as $\frac{1}{2000}$, or are only suggested by doubtful areas, and also in examining the boundaries of defects near the fixation point, and in other ways. They should be used quickly, and the largest object which will show the defect should be selected. Objects between 5 and 15 mm. are useful, and they may be used as small as 1 or 2 mm., but these are only of value for examining the fixation area and its neighbourhood. When using minute tests, whether white or coloured, close to the fixation point, *e.g.*, when determining whether the boundary line goes round it or through it, the fixation object should be small and dull, or, better still, one of the blackheaded pins used for marking out the field should be used as a fixation object. With small visual angles for white and colour exhaustion with restriction of the field is apt to occur in certain cases unless the procedure is rapidly carried out. Large coloured objects are very useful in testing for disproportion, relative hemianopia, detachment of the retina and some other conditions, especially when circumstances render small objects unsuitable, as when central vision is considerably impaired. Darkening of the room in association with the use of both white and coloured tests is a valuable additional method which should always be utilised if there is any difficulty or doubt.

Should neither the perimeter nor the screen examination conducted on these lines reveal any defect, the field may be regarded as normal, at any rate on that occasion. A less detailed examination is inadequate and does not provide the evidence on which a sound opinion can be based. If it is wished to make a more exhaustive search, the visual angle may be reduced by using an object smaller than 1 mm. or, better, by increasing the distance of the screen from the patient. Objects smaller than 1 mm. are difficult to obtain, preserve, and use, while the influence of psychical and other external factors becomes relatively greater.

If defects are found they are analysed by using larger white objects until one is found which is seen in the impaired area, unless, of course, the blindness is absolute. A sufficient number of isopters to show the state of the vision in all parts of the central area of the field is then mapped out.

* The possibility of the presence of narrow scotomata here, due to the trunks of the vessels, should be remembered.

The screen has the advantage that measurements may be made in all meridians at once and marked out during the process, so that the observer sees the field growing under his eye and can rapidly grasp the essential nature of the case. It is most suitable for the examination of the central area while the perimeter is adapted for the periphery. The employment of a combined method when the whole or part of a defect is in the central area leads to saving of time. The test-objects used with the perimeter are carried in to about 20 to 25° from the fixation point, and the blind spot and central field are examined on the screen with test-objects subtending the same visual angles as those used with the perimeter. Thus, if 5-mm. and 1-mm. objects are used at 330 mm. radius, the same isopters may be mapped out in the central field by 30-mm. and 6-mm. objects at 2,000 mm. Although the same visual angles do not always give quite the same fields at different distances this method is sufficiently accurate, and in every way much more efficient and satisfactory than the examination of central defects at a short radius.

Both in perimeter and screen examination it will be found that better results are often obtained as the examination progresses, since the patient acquires facility in concentration and in responding, but in some subjects after a certain point fatigue sets in, and results become uneven and untrustworthy. This should be avoided by testing the eyes alternately for short periods and giving occasional rests. The best work can be done with patients who have had some practice. When patients are difficult more reliable results are often obtained with the screen than with the perimeter; they seem to find the screen examination easier. Sometimes, however, the opposite holds good.

In dealing with small visual angles, such as $5'$ or less, findings showing slight deviations from the normal field need not be regarded as necessarily due to disease, unless associated with characteristic features. Thus, within limits, the shape of the field is more important than its size.

If the screen is marked in concentric circles whose value is known, after the method of Roenne, charting may be done directly during the examination even without previously pinning out the field, and this is no doubt an expeditious method of obtaining a record if the examiner is sufficiently expert. The marking with pins may be very rapidly done, without the patient ceasing to maintain fixation, and has some advantages. The type of field present can be observed as it takes shape, and as its character becomes apparent the method of examination may be modified to suit it, while doubtful parts may be gone over again. If the screen is unmarked the findings are transferred to the chart by a tangent scale, such as Sinclair's rule, aided, if desired, by a protractor on an adjustable stand. During charting the patient is resting with both eyes open. If possible, the figures may be dictated to an assistant, as with the perimeter, thus saving time. It is, of course, not necessary to chart each record separately; if three objects are being used the isopters can be rapidly pinned out and then all charted together. Larger numbers of observations (*i.e.*, more isopters) may be desirable occasionally, but are usually unnecessary in ordinary ophthalmic practice.

Scotometry hardly requires special mention, as it is merely the application of the principle and method of the screen test to the examination of isolated or apparently

isolated defects, and should neither be thought of nor practised as something apart from perimetry. During the systematic examination of the field scotomata are discovered as a matter of course, and should be investigated in the same way with graduated test-objects. It is most undesirable that the peripheral parts of the field should be left uninvestigated in cases in which a scotoma is a pronounced and easily detected feature, though, unfortunately for ophthalmic science, the usual clinical procedure, in the hurry of a busy day, is apt to cause attention to be focussed upon the most intense portion of the defect. A patient, on account of the way he described his symptoms or reads Snellen's or Jaeger's types, may often be known at once to have a scotoma. A rapid test with a single size of object, usually coloured—hat-pin, match-head, etc.—demonstrates the existence of an isolated defect for that test and the matter is settled. It is largely on this account that we know less about these defects than we should, and it is unnecessary to describe here the numerous devices advocated for the detection of scotomata which, however well they may fulfil that function, are usually of little use in enabling us to understand scotomata. When a field containing a scotoma is properly examined by the quantitative method with perimeter and screen, it is not infrequently found that the scotoma is not entirely isolated, but is connected with the periphery by an area of depressed vision. With the screen, for instance, the test-object may be made to traverse a narrow arcuate scotoma from blind spot to periphery without being seen by the patient in a way which would be difficult, if not impossible, with a rotating scotometer. It must be emphasised that not only the position and extent, but also the nature of a scotoma is of importance ; that is to say, its shape and its intensity, especially variations in intensity in different parts, and the steep or sloping gradient of its edge. For the investigation of such details the screen at 2 m. is the best instrument, and an adequate analysis entails the exploration of the defect, projected on as large a scale as is practicable, with graduated test-objects so that the isopters indicating its true character may be properly mapped out.

For the examination of minute central scotomata Marx used the ring type of fixation object. A ring of grey or white paper whose internal diameter corresponds to 3° or 4° may be used. The ring is attached to the screen around the fixation object which is then removed ; the patient fixes the centre of the ring.

In certain cases, such as homonymous hemianopia, in which it is desirable to compare the field of the two eyes in regard to their congruity or incongruity, special care should be taken that the technique with both perimeter and screen is in no way faulty. If the fixation point is too small it may fail to be seen as the examination proceeds ; if too big it may lead to unsteadiness of fixation. The patient must not be allowed to become tired ; the position of his head must be the same during the examination of both fields, and a sufficient number of isopters should be charted to enable a fairly definite conclusion to be reached in respect of sloping boundaries. It is sometimes advantageous to examine the fields alternately with the different tests rather than to test one completely before passing to the other, and to note the order in which the tests were employed.

In homonymous hemianopia and in some functional cases the examination of the

binocular field often provides information of interest and value and should not be omitted if details of the sparing of the fixation area are required.

Difficulties may arise in connection with the patient. A few patients, although they have sufficient central vision, apparently cannot be induced to fix correctly but persist in following the test-object with the eye. In such cases a painstaking endeavour should be made to make the patient realise that he has a blind spot. The test-object, using the screen at 2 m., may be brought over the blind spot area and then turned suddenly edgewise, while he is urged to maintain fixation, and then turned full-face again. If it should prove impossible to map out the blind spot the patient is only suitable for the cruder forms of field examination and accurate results cannot be expected, but patients who at first appear to be impossible subjects often prove capable of giving excellent results once they have been shown that they have a blind spot. Similarly in rare cases it may be impossible to demonstrate a small central scotoma, especially if old, although the symptoms definitely indicate its presence.

Checking Results

A very important feature in the examination, whether by perimeter or screen, is the checking of the patient's responses. This should not be omitted in any case, but is especially valuable when the answers appear inconsistent or improbable. The test-object may be suddenly concealed behind the perimeter arc or elsewhere or may be turned so that its edge presents, or, in colour testing may be made to change its colour almost instantaneously while the interrogation continues. A good plan is to place the object over the blind spot as this verifies fixation also. It will be seen that these manipulations cannot be carried out with a mechanical perimeter, but are easily managed if a thin, double-faced object on a handle is used.

Choice of Test-Objects

When commencing the examination of the field in a case in which the presence of pathological changes is suspected the beginner is faced with the question "What is the best size of test-object to use?"

In all cases several isopters should be examined, *i.e.*, several test objects should be used. To use only one test object is comparable to testing distant vision with a test card bearing only one size of letter.

Adherence to a fixed routine should be avoided. A form of procedure should be adopted which will help the examiner to choose objects suited to the requirements of each case, otherwise time is lost and the patient may become fatigued before the essential characters of the field changes are determined. The general principle is that such objects should be chosen as will best indicate the nature of the case, and elicit and illustrate its clinical features. As a rule good central vision indicates the use of a small object to begin with, and bad vision a large one.

By means of the confrontation test the presence or absence of a gross or moderately dense defect is rapidly ascertained.

The patient is then placed at the perimeter. If no gross defect is present $\frac{3}{30}$ (0.5°)

is a good size of object to begin with as it is large enough to show the normal field and small enough to elicit a defect of moderate intensity. If no defect is found a 1 mm. object is used and, if no defect is found with this, the screen is used with a $\frac{2}{2000}$ or $\frac{1}{2000}$ object, the blind spot being first examined with $\frac{40}{2000}$. Colour tests subtending about a degree, such as $\frac{5}{330}$, or $\frac{20}{2000}$ to $\frac{60}{2000}$ may be used. A few trials with such objects will quickly indicate the nature of the case. If a defect is found its limits of intensity are decided by "straddling." Thus an area defective for $\frac{3}{330}$ is tested with a large object such as $\frac{40}{330}$ and if this object is seen then with $\frac{20}{330}$ and so on until the *largest* object is found which is *not* seen in the area in question. This shows the area of greatest intensity in the defect; the maximum area of the defect is then shown by the smallest object which is not seen. Similarly the seeing area is tested until the *smallest* object which is seen is determined.

It should be remembered that any test-object used on the screen at 2,000 mm.

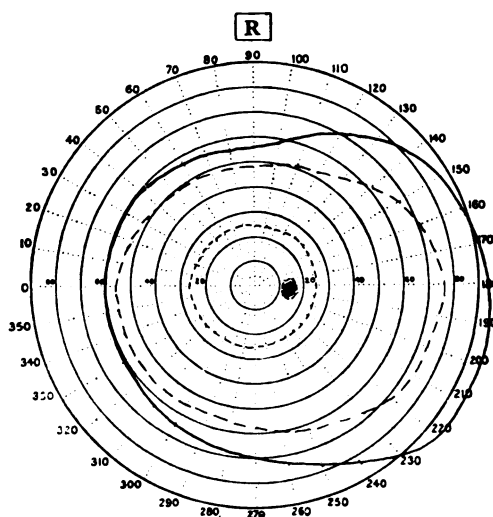


FIG. 18.—EXAMPLE OF A NORMAL FIELD.

Vision $\frac{5}{330}$. No refractive error. Fields for $\frac{3}{330}$, $\frac{3}{330}$ and $\frac{20}{2000}$ in good light. No abnormality disclosed by colour testing or by reduction of illumination.

To indicate the minimum data required to show that a field has been properly tested and found to be normal.

subtends an angle one-sixth of that which it subtends if used on the perimeter at 330 mm.

When small visual angles or colour tests are used the normal position of their isopters should be borne in mind and, in examining the relation of the fields for colour to those for white, care should be taken that the coloured and white test-objects used are as nearly as possible of equal value, i.e., that their isopters in the normal field lie close together.

The perimetrist should have a definite reason for the use of a particular size of object and, while several objects are necessary for an adequate examination, the smallest number should be used, which is sufficient to demonstrate the most important features of the defect.

Symptoms should be explained, if possible; thus, in a case of glaucoma, or of tobacco amblyopia, with R.V. = $\frac{6}{9}$ and L.V. = $\frac{6}{60}$, the charts should show how it is that the vision

METHODS OF EXAMINATION

of the one eye is less than that of the other. A suitable selection of test-objects will show the character of the invasion of the fixation area.

It is this proper selection which makes perimetric work easy and successful, while an injudicious choice may produce results of little value, and render the examination unsatisfactory and inconclusive.

Finally, it may be noted that in order to obtain the full benefit of perimetry in providing its share of the evidence on which diagnosis and prognosis are based, more than one examination is not infrequently required, constituting, in fact, a perimetric study, although in many cases a single sitting will be found sufficient.

To recapitulate the procedure of field testing: The presence or absence of a gross defect is first ascertained by the confrontation method. The periphery is then tested by a visual angle of about 10' or 20' ($\frac{1}{330}$ or $\frac{2}{330}$), and by colour tests, such as $\frac{10}{330}$ or $\frac{5}{330}$ red. If normal results are obtained the screen may be resorted to without spending further time over the perimeter. The blind spot is rapidly mapped out with $\frac{40}{2000}$, and the outer limits for $\frac{1}{2000}$ or $\frac{2}{2000}$ ascertained. Finally the room may be darkened, coloured tests down to $\frac{2}{2000}$ red may be used, or, if necessary, the screen may be removed to a greater distance.

Such an examination can be rapidly completed, especially if charting is not required.

When defects are found they must be examined with suitable tests chosen to illustrate the special features of the individual case, and routine should be avoided. The use of small white test-objects is not to be regarded as a substitute for colour testing.

A purely diagnostic examination including both fields can usually be completed in about twenty minutes, but if careful analysis and accurate charts are required more time is necessary, or even a second sitting.

Special attention should be paid to the following points:—

The result of the examination by the confrontation method should be used as a guide to the part of the field upon which attention is to be concentrated and to enable all unnecessary work to be avoided so that the examination can be rapidly completed. It is the detection or exclusion of defects and not the precise measurement of any part of the field that is of importance. Features requiring more exact investigation may be dealt with as circumstances indicate. The perimetrist should always remember that it is the *kind* or *type* of defect that is of importance and not its size.

The patient should be made perfectly comfortable in every way. If the examination is prolonged he should be allowed to rest occasionally and care should be taken that in these intervals relaxation is complete.

If the patient should become tired it is better to postpone the remainder of the test.

(4) Special Methods—

Perimetry in Dim Light

Reduction of the illumination is a very important and valuable method of modifying the stimulus and is specially useful in clinical diagnostic work. This method is easy to apply by simply drawing down the blinds. Moderate reduction of illumination does not cause any notable depression of the field in a normal case, at least until the light is so dim that the fixation object is hardly visible to the examiner himself, but in pathological

conditions defects are accentuated so that relative defects of low intensity can be more easily demonstrated. In conditions affecting the outer layers of the retina useful information may be gained by testing the field in a dim light when defects, especially relative blue blindness, not well brought out otherwise, may be disclosed or accentuated. Also in conduction defects of very slight intensity positive findings may be obtained which could not otherwise be elicited, since impairment of the light sense is an early symptom of diminished nerve conductivity. When no defect can be elicited in ordinary illumination the light may be reduced until a considerable contraction of the field is produced. Test-objects of 2 to 5 mm. in white, red or blue may then be used at distances of 2 to 4 m. in the search for faint defects of the central area of the field. It may be desirable to make the room so dark that an object such as $\frac{2}{2000}$ white cannot be seen by the examiner.

The method is clinically useful in some cases in that it enables relatively large test-objects to be used, and demonstrates the qualitative nature of the defect. Without special means it is impossible to measure exactly the amount of light used; this is, however, not necessary and the illumination may be adequately gauged by the effect on the observer's own visual perception.

The Examination of Peripheral Vision in the Dark-Room

1. THE EXAMINATION OF THE SENSE OF LIGHT-PROJECTION

This is the most commonly employed and most useful form of examination of peripheral vision in the dark, a method specially adapted for the investigation of cases in which ordinary perimetry is impossible owing to bad vision depending on opacities in the media. The patient is seated and asked to look straight before him, if his vision is very poor he may be asked to look where he hears the voice. A weak beam of light, conveniently obtained by means of an ophthalmoscopic mirror, is directed from a peripheral part of the field on to the eye under examination, the other being securely covered, and the patient is required to indicate from which direction the light proceeds. No other light should be allowed to fall upon the eye. The process should be repeated, using coloured glasses placed before the eye.

In this way the whole field is examined, including the central area. The test finds its application usually where the state of the iris, lens or vitreous precludes the usual methods of examination, and is chiefly used in examining cataract cases.

The indications afforded by this method may be shortly referred to here. Should the perception of light be good and its localisation accurate, it may usually be considered that the retina and optic nerve are healthy and vice versa. There are, however, certain exceptions. Vision is sometimes retained to a certain extent in a detached area of the retina and in other conditions without marked interference with light projection, and thus hopes based on operation may be disappointed. On the other hand, in cases of retinitis pigmentosa the peripheral light projection may be bad, and yet the patient may be benefited by removal of cataract as sufficient central vision may still remain to be of value.

2. DARK-ROOM PERIMETRY

Perimetry in a dark-room with luminous test-objects has been advocated for some years. The appliances have already been discussed. Practical considerations seem likely to prevent this method being widely adopted as a clinical procedure. Unless the patient is confined for about forty minutes in darkness the process of adaptation is not complete and stationary, and confusing factors may be introduced. Although this method has been advocated as specially useful in certain conditions, such as detachment of the retina, it has not yet been shown to produce more useful results than those obtained by the use of small visual angles in daylight, or in reduced illumination with the ordinary apparatus.

According to Lythgoe (269) during dark adaptation colour sensation is diminished and the power of discriminating hues reduced.

In all its various methods, though not always in the same degree, perimetry, it is essential to recognise, is a highly subjective form of examination; an examination of the patient's sensations as described by himself in answer to questions put to him by the observer. The results obtained depend on the influence of several factors of which the state of the visual pathways and centres forms only one, though, of course, nearly always the most important, and their value varies with the ability, experience and discrimination of the perimetrist in distinguishing and eliminating the less essential features. The scope of perimetric examination is limited on the one hand by the nature of the tests involved, and on the other by the capacity of the subject to respond to them. Demands are made upon attention, concentration, intelligence and reaction time as well as upon vision, by tests which, in comparison to tests for a physical problem, are relatively crude, and, in addition, suggestive influences, unless carefully guarded against, are constantly, if unconsciously, passing from the examiner to the subject.

In perimetry the personal equation and training of the clinician are of great importance and no form of ophthalmic examination makes greater demands upon him. It is for this reason that different observers will often obtain different results in the same case.

Perimetric charts, therefore, should not be regarded as conveying a mathematically accurate and precise expression of the state of peripheral vision, but rather as indicating the kind and degree of defect which is present, and require to be interpreted liberally with a knowledge of what perimetry entails and of the special nature of each case. What is really essential is that such charts should be comparable one with another. It has been pointed out that our methods of examination involve various errors, but if the same method under the same conditions is applied to every case, and to every examination in the same case, the influence of such errors will be negligible as far as clinical ophthalmology is concerned.

The beginner should endeavour to acquire a good technique by the study of cases of glaucoma simplex, in which fixation is good. Experience and facility with screen work should be acquired by making detailed studies of the normal blind spot. He may then proceed to the examination of the hemianopias and of tobacco amblyopia.

C.P.

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CHAPTER IV

PHYSIOLOGY OF THE VISUAL FIELD IN RELATION TO CLINICAL PERIMETRY

IN applying tests of the kind suggested to the examination of the field it is well to remember some of the more essential features of the response of the visual nerve mechanism. We have seen that perception is less acute at the periphery of the field than in the centre, and that our estimate of the value of different parts of the field is based upon what can be perceived there during fixation of the eye.

In perimetry the image of a white object upon a black background falls upon the retina, and, as a result, the power of perceiving white in that area of the retina is rapidly used up, so that, if fixation is steady, the white object will gradually fade away entirely. At the same time, however, the surrounding retina is becoming more efficient for the perception of white, so that if the direction of fixation is slightly altered the white object is immediately seen again and very brightly. If fixation were absolutely steady and all objects in the field stationary, they would gradually disappear—those peripherally situated first, and finally the object fixed—but would reappear as soon as a slight movement of the eye caused their images to fall upon an adjoining part of the retina. This can easily be demonstrated by placing one or more large white objects on the 2-m. screen at different distances from the centre, at which a 5-mm. object is attached. Steady fixation of the latter is quickly followed by the disappearance of the eccentrically placed objects in the order of their distance from the centre. They reappear at once if fixation is altered. (See also Tscherning 415.)

Under normal conditions the continuous perception of objects is made possible by a slight rapid movement of fixation over the surface of the object regarded, excursions up to about 5' in extent, with occasional larger movements, being made in all directions. As a result the area of the retina which is used to receive the image of the object fixed is larger than the image, and thus, while some retinal elements are photochemically active, others, which will be brought into use the next instant, are recuperating and becoming prepared for the efficient reception of the image. The movement of the visual axis moves the whole field and influences the perception of peripheral objects in the same way. This physiological unsteadiness or travel of fixation has only a minimal effect on the normal limits of the field or the boundaries of defects as mapped out by the perimeter or screen. Were it not so all defects would have indefinite edges, which is contrary to clinical experience. The explanation is, no doubt, that the mental impression, formed from retinal images and projected into space, is stationary although the retina itself moves.

Similarly the image of a coloured object, red for example, on a part of the retina uses up the power of perceiving red and increases the power of perceiving its complementary colour, green, at that spot, while the surrounding retinal elements become favourably disposed towards the perception of red and unfavourably towards that of green.

The fixation object should, therefore, not be too small and should have a black centre. If a very small object is required for the examination of the immediately central part of the field it should be replaced by a slightly larger one when that part of the examination is completed. The test-object, white or coloured, should be kept moving slightly. Seams or marks on the perimeter arc or on the screen are of little or no consequence if not sufficiently numerous or large to alter the character of the background.

These physiological processes in the retina appear to have little influence upon the results of perimetric examination under ordinary circumstances. The breaking down and building up processes mutually support each other, and the retina does not get tired and refuse to function. Variations in response are due to variations in central rather than retinal function, and the terms "retinal fatigue" and "retinal asthenopia" have probably a much more restricted significance than is frequently attributed to them. A patient's field often becomes wider for a small object after he has been examined for some time, when his interest and attention are aroused, and it soon becomes smaller when he—not his retina—becomes tired. A slightly subnormal power of concentration with flagging attention leading to an apparent oscillation or stammering, as it were, of perception, may be the explanation of certain concentric ring scotomata noted by some observers. The weaker the stimulus the more likely will such phenomena be to appear, hence the usual peripheral situation of physiological ring scotomata.

This aspect of the physiology of vision is of special interest in connection with functional field changes into which the element of suggestion enters so as to exaggerate and distort normal conditions.

PART II

APPLIED PERIMETRY

INTRODUCTORY

THROUGHOUT the sphere of medicine experience has taught us not to rely on any one sign or symptom as affording conclusive evidence in diagnosis and prognosis. Such a rule, while applicable in varying degrees to different cases, is always of value in drawing attention to the desirability of basing our views upon a complete clinical picture in which all the features are presented in proper proportion and perspective.

Obscurity of vision with apparently clear media, thus presumably depending on some affection of the nerve mechanism of sight, constituted the "Gutta serena," "Black cataract" or Amaurosis, and, when incomplete, the Amblyopia, of the older writers. Mackenzie, whose notes on hemianopia are still not unworthy of our attention, regarded this condition as "merely a peculiar variety of amaurosis," and gives a list of no less than fifty-three forms of amaurosis under the headings, Retina, Optic Nerve, and Brain. He knew that amaurosis was "a mere symptom" and that treatment must be directed towards the cause. In regard to diagnosis, however, he is almost silent, only pointing out that it may be easily confused with glaucoma and emphasising the importance of distinguishing incipient amaurosis from incipient cataract. Referring to the gradual loss of vision in the latter he says, "Whereas, in amaurosis, the attack is often sudden, and being partial is described as a dark spot or spots occupying certain parts only of the field of view. . . ." This and some remarks in connection with hemianopia seem to be his only references to the field of vision.

Such was the position a few years before ophthalmoscopy and perimetry had come to the assistance of the ophthalmic surgeon, whose unaided gropings, when all is considered, had been really extraordinarily successful. The fourth edition of Mackenzie's book appeared in 1854. About the same time the ophthalmoscope began to be used and Von Graefe drew attention to the importance of the examination of the field of vision. As a result, the term "amaurosis" has become merely a synonym for blindness without obvious cause, and Mackenzie's fifty-three varieties have been duly assigned to their proper positions, while, in addition, the prognosis as well as the diagnosis of many previously obscure conditions has been enormously advanced.

It is, then, mainly in regard to conditions which were formerly grouped under the terms "amaurosis" and "amblyopia," including, as in all probability they did, many cases of undiagnosed glaucoma, that the evidence obtained by perimetry forms an important, sometimes indeed the chief, feature in the composition of the clinical picture. Viewed in its proper relation to other methods of investigation, perimetry is indispensably associated with the subjective examination of the eye in the same way as ophthalmoscopy is with the objective. While its most striking and most valuable advantages are in connection with diagnosis, especially of affections of the visual path and centres, evidence throwing light upon the normal anatomy and physiology of these structures is

constantly being provided. Much information has also been gained with regard to the mechanism of the production of symptoms and the anatomical and pathological conditions underlying many visual affections, enabling their prognosis to be placed upon a surer footing and their treatment upon a more rational basis.*

In clinical work the function of perimetry is to discover the cause of depression of vision not adequately explained by other methods of examination, and also to measure the degree and progress of the morbid process. The conditions in which it is of value may be grouped somewhat as follows :—

- (1) Those in which the media are clear and the fundus oculi normal.
- (2) Those in which ophthalmoscopic evidence is available but requires amplification and explanation.
- (3) Those in which the media are partly obscured, making ophthalmoscopic examination impossible or inconclusive and in which disease of the retina or visual path is suspected or should be excluded.

It is especially valuable in cases in which visual impairment is apparently the only abnormality.

The situation and extent of the visual path in the brain indicate the importance of perimetry to the neurologist and the cranial surgeon, and the visual symptomatology of intracranial lesions, a territory previously largely unexplored, is now being actively investigated by modern perimetric methods.

* Unthoff (418) wrote in 1900: "In no nerve territory is functional testing elaborated to such an extent as in connection with the optic nerve: . . ."

The significance of perimetry in diagnosis and prognosis was foreseen by Berry (33, p. 117), who wrote in 1886: ". . . But it is evident that there are many points of diagnostic importance to be gathered from an examination of the field. . . . But there can be little doubt that in the thorough examination of the relative as well as absolute functional activity of all parts of the retina we possess a means of immense value in the localisation of intracranial disease. But it is not merely as a method of diagnosis that this examination deserves attention; in many cases it affords the most delicate means of ascertaining the course taken by any disease, especially when of intracranial origin."

CHAPTER V

THE PATHOLOGICAL FIELD

IN conditions of disease which give rise to impairment of the visual function this impairment is manifested as an alteration in the visual field. Regarding the field as a circumscribed area, such alterations appear as reductions of the extent of the area or *contractions*, or gaps or voids within the area or *scotomata*.

Reverting to the simile of a hill of vision surrounded by a sea of blindness, we can imagine the hill sinking into the sea so that its circumference becomes reduced, or we

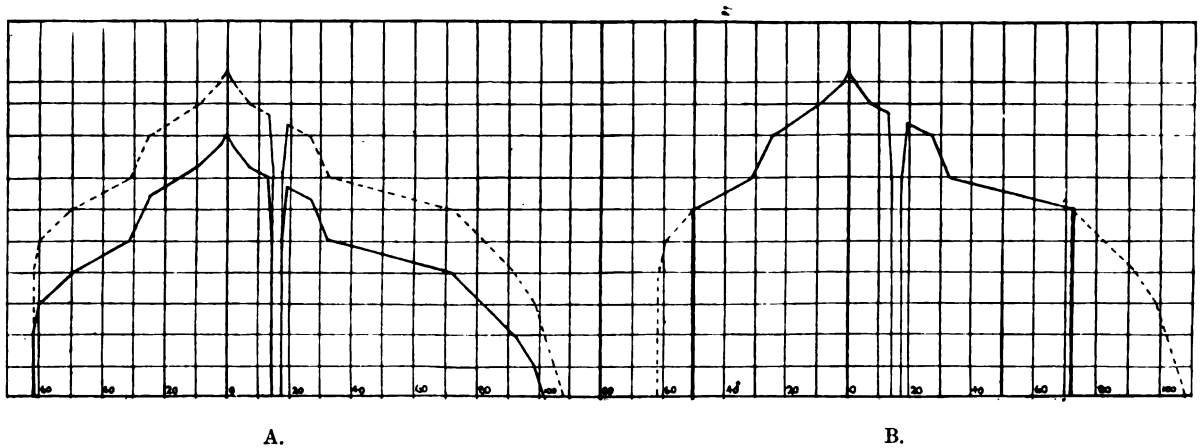


FIG. 19.—CONCENTRIC CONTRACTION.

Diagrammatic scheme illustrating the difference between contraction and depression. The broken line indicates the outline of the normal field. Scale of values as in Fig. 2.

A represents the depression type of contraction, B the true contraction or coast erosion type.

In A the field is reduced in extent all round except on the nasal side and would be reduced there also were the depression sufficiently great. All the isopters are affected. The greatest contraction is present where the slope of the field is most gradual, about the level of the 2000 isopter. If A were tested with a single large object only a small contraction at the temporal periphery would be found and the true state of the field would remain undetected. The real nature of the condition can only be determined by the use of several test-objects.

In B the field is reduced in extent all round, including the nasal side, but only the peripheral isopters are affected. The field is not depressed but genuinely contracted. Here also several test-objects are required to establish the nature of the condition, small ones to show the integrity of the central part and large ones to demonstrate the peripheral restriction.

can imagine the sea washing away the land while the remainder of the hill retains its original form. There are, therefore, two possible types of change, the depression type and the coast erosion type, and these may be combined. When depression occurs the field boundaries become constricted, hence the field is often called "contracted." The constriction is less where the edge is steep (cliff) and greater where it is sloping (flat beach). A slight depression is therefore most easily detected by examining the circumference at a part where the edge is sloping.

A depression may occupy the whole or any part of the field and may be of any shape, size, or depth, with flat, crateriform or uneven floor, and sloping, steep or precipitous edges. It may occur suddenly, rapidly, or slowly, and may increase, fluctuate or diminish. These features constitute its *characters*.

Pure peripheral loss with the remaining part of the field normal—the coast erosion type—is rare, if indeed it ever occurs, but is not infrequently closely approached in certain forms of disease.

In order to understand these field changes we must, therefore, think of the field as a surface which falls and rises, and not as an area which contracts and enlarges.

The nature of the field defect is shown by its characters, which should always be systematically examined.

These are: (1) Position; (2) shape; (3) size; (4) intensity; (5) uniformity; (6) margins; (7) quality; (8) behaviour or course.

As the field of vision is always schematically represented on a chart, it will be convenient to adhere to the current terminology of field changes, although this is based on the idea of a contracting and expanding area.

Two main groups may be distinguished: the *contractions* and the *scotomata*.

I. Contraction of the Field

Position and shape may be considered together.

(1) *Position* and (2) *Shape*.—A contraction or restriction is a defect which affects the periphery. When the contraction becomes extreme, the central area is involved. Contraction may be concentric or local.

Concentric Contraction.—In concentric contraction the whole periphery is more or less equally restricted so that the field is reduced in size without much alteration in shape.

Concentric contraction is, as has already been stated, nearly always a general or widespread depression of the field rather than a mere restriction of its peripheral limits. Such a depression appears as a concentric contraction if only one peripheral isopter is demarcated; if several are determined the true state of the field is immediately disclosed. The relationship of the isopters (Fig. 1), which lie close together at the extreme periphery, but are more widely separated in the interior of the field, especially on the temporal side, shows that a low degree of general depression does not produce a demonstrable contraction of the peripheral but only of the internal isopters. The early stages of general depression are, therefore, most easily detected by examining the field where its slope is most gradual (see Fig. 2), *i.e.*, by using a test which corresponds to an internal isopter such as a colour test or a small visual angle for white. The area immediately to the temporal side of the blind spot is especially weak, so that in a slight degree of depression the isopter for $\frac{1}{2000}$ falls to the nasal instead of to the temporal side of the blind spot.

When depression is sufficiently advanced to produce an obvious restriction of the peripheral isopters the contraction is greater on the temporal side, since the gradient slopes less steeply there than on the nasal side. Before the stage is reached at which the nasal isopters also begin to show contraction the limitation may appear to be confined to the temporal field simulating a temporal, or if bilateral, a bitemporal “contraction.” This is commonly found when too large a test-object has been used and is, of course, not the same thing as bitemporal hemianopia. Uniform depression suggests the probability of a functional as against an organic cause.

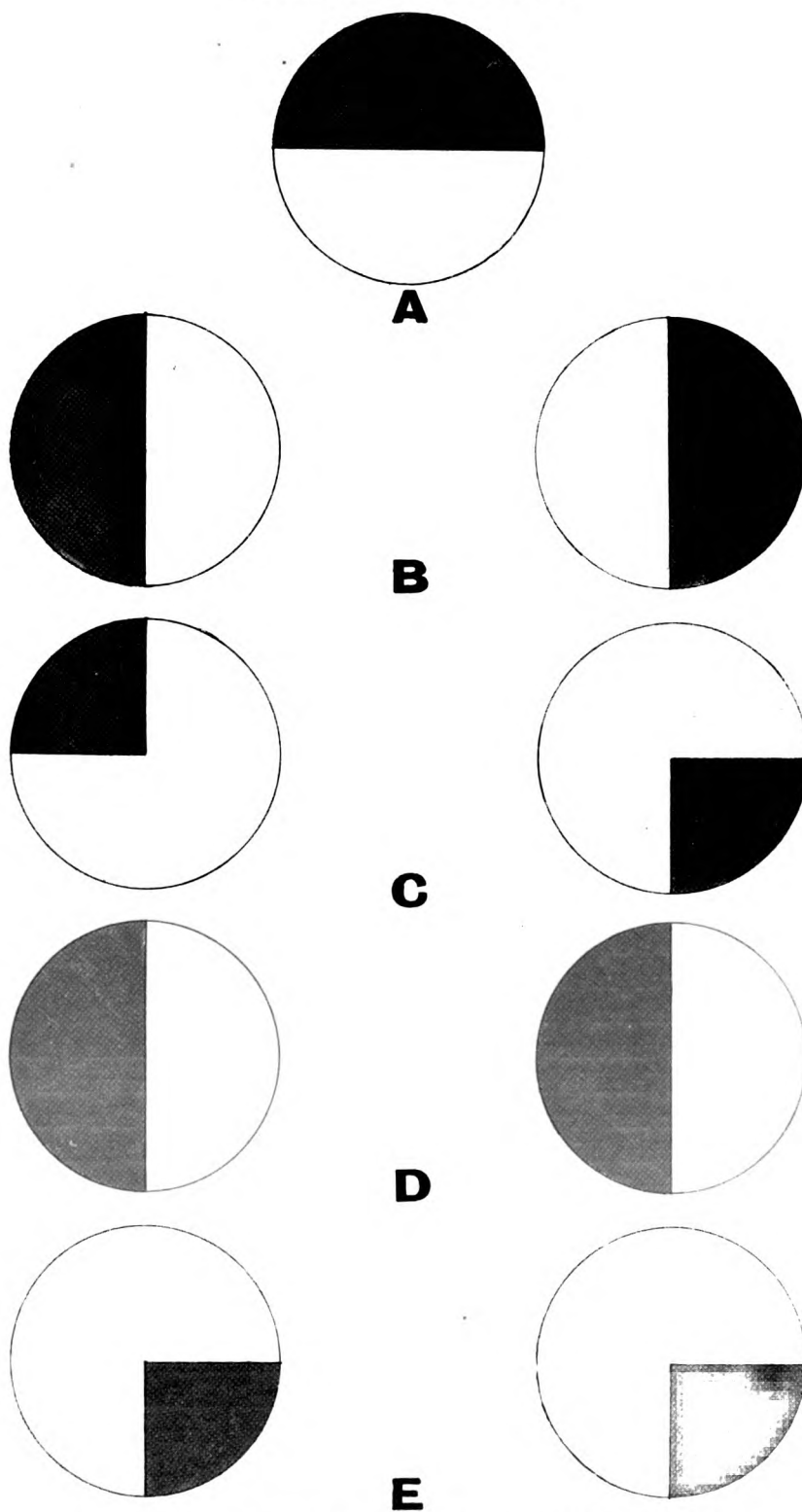


FIG. 20.—THE DIFFERENT FORMS OF HEMIANOPIA (1) (DIAGRAMMATIC)

A, Horizontal or altitudinal. B, Bitemporal. C, Bitemporal crossed quadrant. D, Homonymous. E, Homonymous lower quadrant.

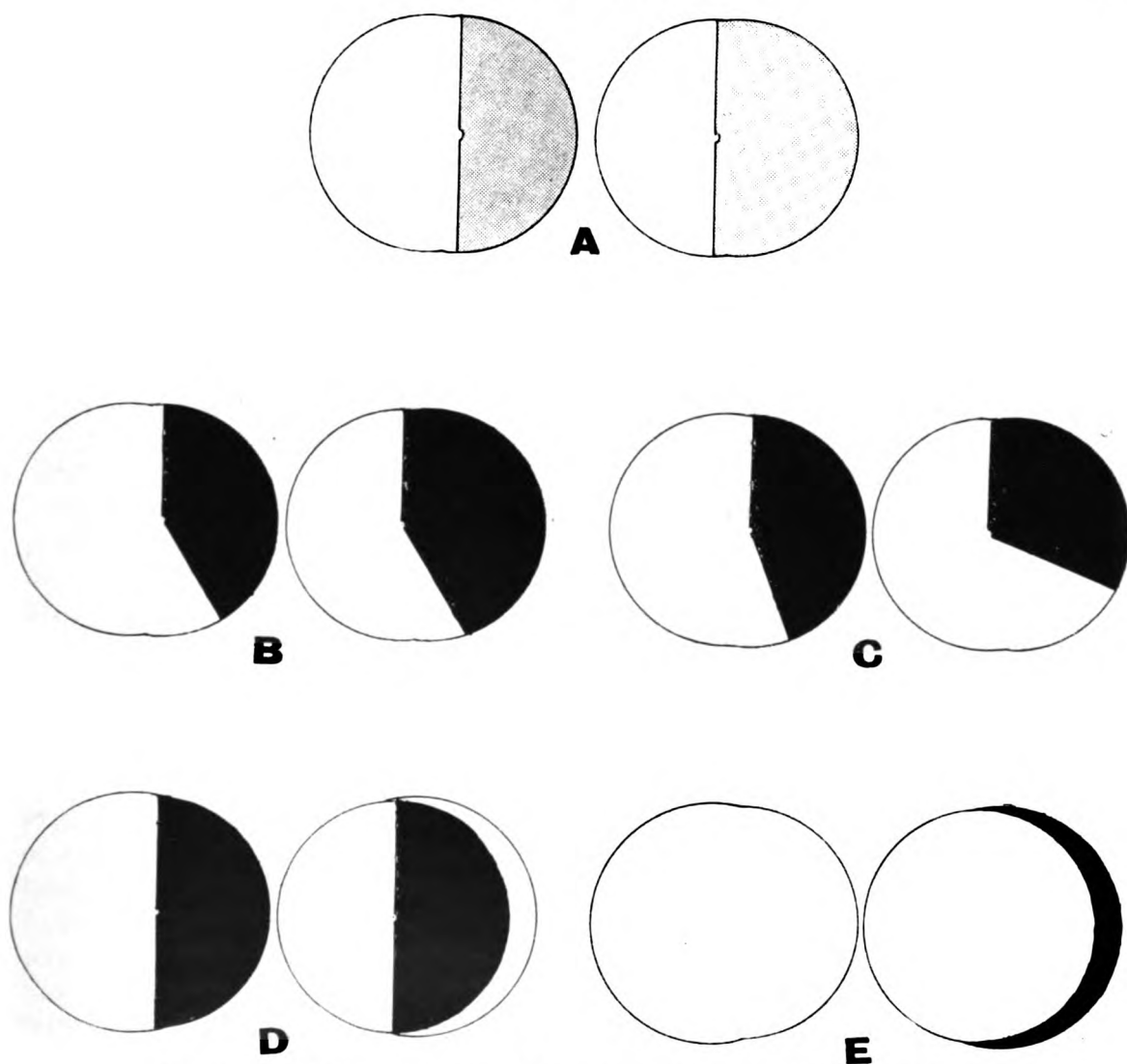


FIG. 21.—THE DIFFERENT FORMS OF HOMONYMOUS HEMIANOPIA (2) (DIAGRAMMATIC).

- A. Relative right homonymous hemianopia, complete.
- B. Incomplete absolute right homonymous hemianopia.
- C. Incongruous homonymous hemianopia.
- D. Absolute right homonymous hemianopia with sparing of the temporal crescent.
- E. Absolute right temporal crescent loss alone. (Although there is no hemianopia in the literal sense this condition belongs to the group of homonymous hemianopias.)

As a rule, the depression is not uniform, but greater at one part or another. When it is entirely or mainly peripheral true peripheral contraction (coast-erosion type) is present, but this is not as common as the familiar use of the term suggests.

A special form of concentric contraction is the "tubular" field. The depression is severe and involves the whole field with the exception of an area surrounding the fixation point, producing a great and often extreme contraction. The linear extent of the field is found to be the same at whatever distance the examination is carried out, so that

the projection is apparently cylindrical instead of conical. Such fields are obviously of functional origin. In other cases of an allied nature the depression—and with it the concentric contraction—varies in degree as the examination proceeds, giving rise

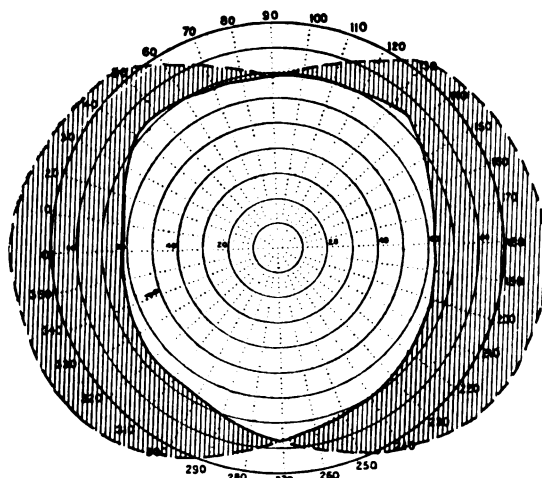


FIG. 22.—THE EFFECT OF BITEMPORAL HEMIANOPIA UPON THE BINOCULAR FIELD. THE SHADED AREAS INDICATE THE LOST UNPAIRED PORTIONS. THE UNSHADED AREA REPRESENTS THE NASAL FIELDS OF THE TWO EYES PLACED SIDE BY SIDE AND HAVING ONLY UNIOCLAR VISION.

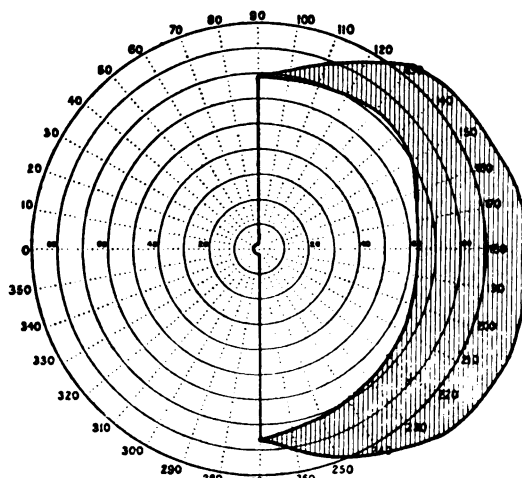


FIG. 23.—THE EFFECT OF A LEFT HOMONYMOUS HEMIANOPIA UPON THE BINOCULAR FIELD. THE SHADED AREA INDICATES THE UNPAIRED PORTION OF THE RIGHT FIELD, IN WHICH UNIOCLAR VISION IS RETAINED. THE UNSHADED AREA REPRESENTS THE NASAL HALF FIELD OF THE LEFT EYE SUPERIMPOSED ON THE TEMPORAL FIELD OF THE RIGHT EYE. THIS AREA, INCLUDING THE FIXATION AREA, HAS THEREFORE BINOCULAR VISION.

to the spiral field or to a star-shaped field respectively when the meridians are tested in consecutive or alternate sequence.

Depression of the central field alone with apparently intact periphery is demonstrable as concentric contraction of the central isopters only. This is a little recognised defect which might perhaps be more accurately classed as a form of scotoma.

In widespread depression, the existence of practically full vision ($\frac{5}{6}$ to $\frac{6}{6}$) by Snellen's type is no proof that foveal vision is unaffected. The central peak of visual acuity may be considerably lowered before vision is reduced below the $\frac{5}{6}$ level.

In irregular concentric contraction the depth of the depression varies in such a way that the isopters are no longer approximately circular, but show an indentation at one part or another. When the depression in the less affected part of the field is minimal this type of defect merges into the local contractions.

Local Contraction.—In local contraction only one part of the field, including the periphery, is affected. An important form is the *sector defect*. Sector defects are depressions roughly or exactly defined by two radii of the field. The sector may be only a narrow wedge or may occupy a large part of the field. Two separate sector defects are sometimes present.

Sector defects bounded by vertical or horizontal diameters of the field are *hemianopic* defects. A quadrant defect is bounded by vertical and horizontal radii. When the boundaries are not straight the hemianopic defects are irregular. The term "hemianopia" (*syn.* hemiopia, hemianopsia) is used to indicate a defect occupying half the field, usually a vertical half. A unilateral hemianopia is a hemianopic defect of one field only, and, therefore, of subchiasmal origin, though, if partial and limited to that part of the unocular field which is unpaired in the binocular field, it may be due to a suprachiasmal interference.

It is preferable to reserve the term "hemianopia" for bilateral field defects produced by a single lesion which must be chiasmal or suprachiasmal in situation. True hemianopia is, therefore, a defect of the binocular field. It is evident that bilateral, symmetrical, subchiasmal lesions of the visual path can produce a somewhat similar state of the binocular field, but this would be a combination of independent unilateral hemianopic defects rather than a true hemianopia. Examples of defects of this kind are found in the apparent bitemporal hemianopias which may sometimes be found in gross papilloedema or in tobacco amblyopia and some other conditions. It should be recognised that true bitemporal hemianopia means a definite type of defect, which exhibits not only impairment of the temporal fields, but also the characters of chiasmal interference.

Hemianopia is homonymous, right or left, when the corresponding half-fields of both eyes are affected, and heteronymous, bitemporal or binasal, when both outer or both inner half fields are involved. The binasal form is not a true hemianopia.

Defect or loss of the upper or lower half-fields is termed *altitudinal* or *horizontal hemianopia*. This may be superior or inferior and unilateral or bilateral, and in the latter case symmetrical or crossed (one upper and one lower half-field defective). It is very rarely a true hemianopia, but usually merely a wide sector defect.

When a quadrant of each field is affected, quadrant hemianopia or tetrantanopia is present, and may be homonymous, bitemporal or binasal, and upper, lower, or crossed (one upper and one lower quadrant) in each case. Hemianopic defects are incomplete or partial when the whole extent of the half or quadrant concerned is not affected, and they may be relative (hemiambyopia or hemiachromatopia) or absolute.

Congruous homonymous hemianopic defects are equal in the two fields and super-

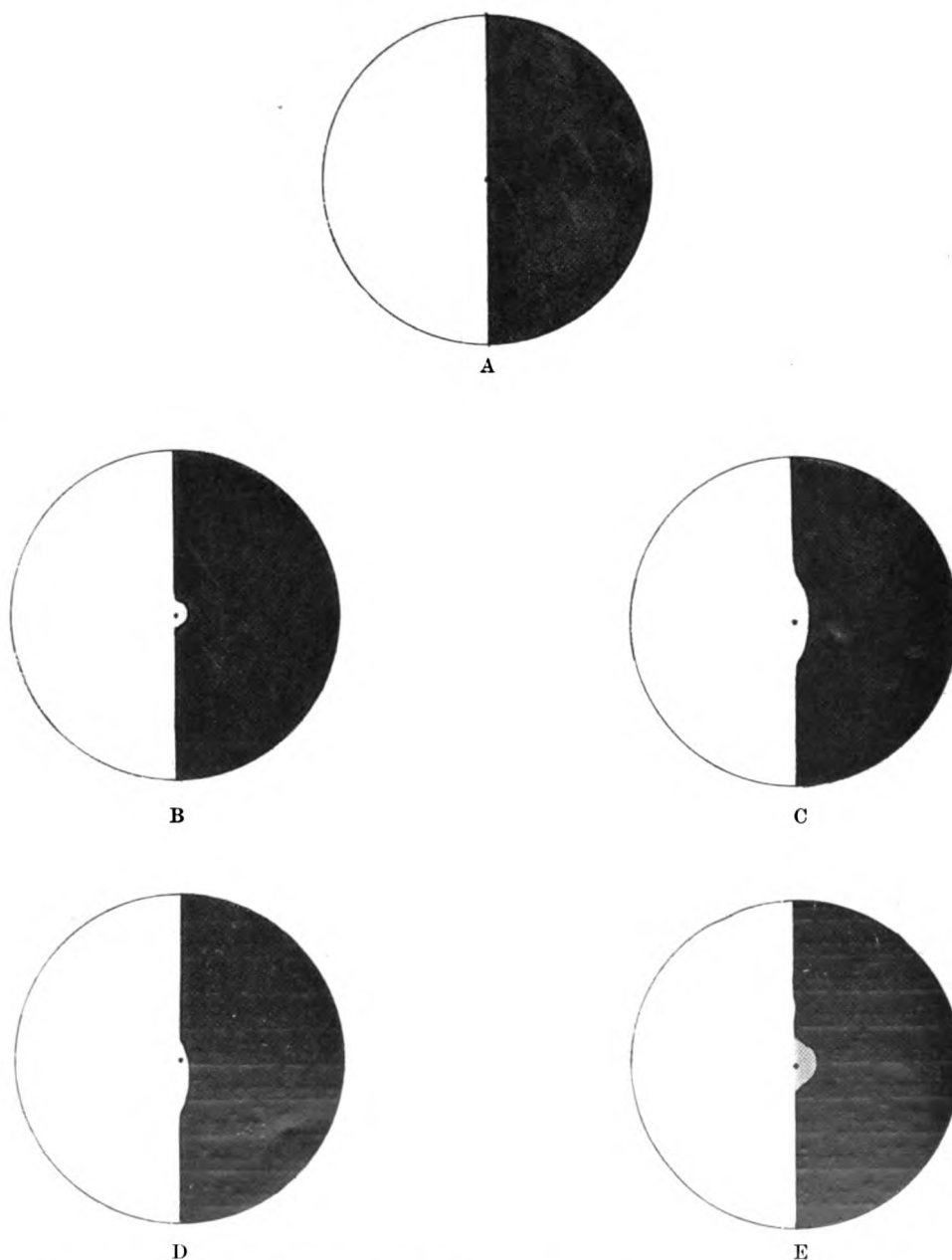


FIG. 24.—RELATION OF DIVIDING LINE IN HEMIANOPIA TO FIXATION AREA (DIAGRAMMATIC).
 A, Divided fixation area. B, C, D, Different forms of sparing of the fixation area. E, Relative sparing.

impossible, forming a single defect of the binocular field; other homonymous, hemianopic defects which are unequal either in extent or intensity are *incongruous*.

Since the temporal is larger than the nasal field all complete homonymous hemianopias are incongruous in extent in respect of the temporal crescent. Incomplete homonymous hemianopia may also exhibit this form of incongruity. That is to say

incongruity may be present on account of the unpaired part of the superimposed fields though the defects of the paired parts are identical in onset, intensity, and extent. The only field changes which are congruous in all respects are homonymous hemianopic defects affecting the paired parts of the fields only, with sparing of the temporal crescent. Congruous defects, and those in which the incongruity refers only to the temporal crescent, are of suprageniculate origin while incongruous defects are due to sub-geniculate lesions.

Corresponding sectors may be affected in both fields and may be either congruous or incongruous. The former are congruous homonymous sector defects and may be regarded as a variety of incomplete hemianopia in which the defect is not bounded by the vertical meridian at any part.

The effect of bitemporal hemianopia upon the binocular field is less than that of the homonymous form, since only the temporal unpaired portions of the binocular field are lost; the central paired portion is retained, although vision within it is now only uniocular. In homonymous hemianopia one-half of the binocular field is lost, but binocular vision is retained in the paired portion of the remainder. (Figs. 22, 23.)

The line of the vertical meridian of the field which separates the blind from the seeing portion may divide the fixation area, or may pass round it so as to include it in the seeing field. In this case the fixation area is *spared*.* The amount of the sparing varies from a mere trace to several degrees. In rare cases the dividing line may be altogether several degrees to one side of the vertical meridian (overshot fields), or it may lie along a slightly oblique meridian. The demonstration of the exact relationship of the dividing line to the fixation area usually requires very careful examination with the screen using delicate tests (see p. 252).

The seeing half-fields may be normal or may be affected in various ways.

Unless homonymous hemianopic fields are carefully tested with test-objects of different sizes it may not be possible to elicit the true nature of the changes. In homonymous hemiamblyopia the temporal periphery may be blind and the slight depression of the affected homonymous half-fields may escape notice unless definitely sought for. Similarly a defect confined to the temporal crescent, if of slight degree, may very easily escape observation unless the examination is adequate.

Bilateral homonymous hemianopia is present when both right and left homonymous hemianopia exist together. There is usually a small central field remaining corresponding to the bilateral sparing of the fixation area.

Irregular sector defects occur in various forms. They merge into and include those known as "re-entering angles." Many of these changes are wedge-shaped defects, the apex being at the blind spot instead of near or at the fixation point. These are known as nerve fibre bundle defects since they depend upon the involvement of a group of adjacent nerve fibres. Their extension to the periphery technically separates them from scotomata. A defect of this kind which has one boundary on the nasal horizontal meridian exhibits the *nasal step* (Roenne 321).

(3) The *Size* or extent of field defects varies within extreme limits and corresponds

* Usually, but wrongly, called "sparing of the macula."

to the number and distribution of the nerve elements affected. A small lesion in a concentrated part of the visual path may produce an extensive alteration in the field.

In all forms of depression or contraction it is the shape rather than the size of the field which affords evidence of value in diagnosis.

(4) The *Intensity*, density, or saturation of defects varies from absolute blindness to a barely demonstrable impairment of vision. The patient may be quite unaware of the presence of a defect of low intensity, and such defects often escape observation unless the field examination is exhaustive.

A defect is absolute when no perception of light exists in the area concerned. This should be proved by the use of sufficiently strong stimulation to exclude any degree of intensity short of complete loss of response. It will then be found that really absolute defects are not as common as a less exhaustive test might lead us to believe. When the defect is small it is very difficult or impossible to demonstrate its intensity accurately, as adjoining nerve elements may be stimulated by the test employed. In such cases the visual angle used should be stated. Thus a small defect may be blind for all visual angles up to $\frac{10}{330^\circ}$ but not for larger angles, and though it is probable that such a defect is absolute, this cannot be proved.

Relative defects are defects which are not absolute. The frequency with which relative as opposed to absolute defects are found depends on the method of examination; when this is thorough, it will be found that many defects hitherto regarded as absolute are really relative.

Colour defects or dyschromatopias of whatever type, except, of course, congenital colour blindness, are relative defects and, unless slight, are associated with some demonstrable loss of perception of white. Examples in which the smallest white test-object practically available is seen are often met with, and the quality of a relative defect is more satisfactorily determined by testing with colour. Colour defects, whether for one or more colours, are simply part of a general lowering of visual response dependent on imperfect stimulation of the visual perceptive centres and have no individual specific significance in relation to the nature of the causal lesion. (See Berry 34.)

An interesting and important feature in the intensity of defects is the relation of the loss for colour to that for white. In the normal field the isopters for colour are comparable with those for white, except perhaps at the extreme periphery. Owing to the size of test-object commonly employed, colour isopters are usually internal isopters, and, therefore, the colour fields are found to be smaller than those for white. Since depressions of the field produce their effects more obviously upon the internal isopters colour defects may be found to precede or to exceed the field changes found with white tests. If the colour test used gives an isopter lying normally considerably within that corresponding to the white test, an apparently initial or excessive loss for colour may be discovered, quite apart from any special impairment of colour perception. A genuine difference between the colour loss and that for white is, however, a common and characteristic field change. There may be a relative yellow-blue amblyopia (see (7) "quality," p. 63) in impairment of the photochemical apparatus. Vision for colours may be seriously affected in association with good vision for white, or, in other cases, vision by Snellen's

types may be reduced while that for colour tests remains relatively good. In conduction defects a true excess of red-green defect over that for white is common, or under other conditions the loss for colour may be in accordance with that for white. In the former case *disproportion*, in the latter *proportion* (Roenne 327) is present. To demonstrate this feature, it is obvious that the test-objects, to give comparable results, should be such as give isopters which lie close together in the normal field. Roenne states that the field for red $\frac{10}{300}$ (114') should be approximately equal to that for white $\frac{10}{2000}$ (17') (*i.e.*, some 5 to 10° within the usual limits of the normal field). Should the red field be slightly smaller than this, say, as small as the field for white $\frac{5}{2000}$ (8') (10 to 20° within the full boundary), no special inference need be drawn, but if the red field is markedly smaller than that for white "disproportion" is present and indicates an actively progressive process. "Proportion" between the fields for red and white indicates that the condition is relatively stationary.

Failure to recognise a large red object in a defect in which small white objects are perceived may suggest that the defect is present for colour only. Such defects are, however, examples of disproportion since the impairment of vision for white can be demonstrated if a sufficiently small visual angle is employed.

(5) *Uniformity*.—The intensity, *i.e.*, the depth of the depression or the degree of visual loss, may be the same all over the altered area, or it may differ in different parts. There may be areas of greater or lesser intensity within its limits. The determination of the composition of the defect by the use of a series of test-objects constitutes the *analysis* of the defect and is an essential part of perimetry and frequently provides the key to the nature of the underlying condition.

In estimating the degree of functional failure in the area of a field defect it should not be forgotten that the normal visual efficiency is physiologically less towards the periphery of the field. The effect of a uniform degree of loss of physiological activity over the area of the defect would therefore be more easily demonstrable in a part towards the periphery of the field and might be elicited only with difficulty in a more central part. For example, only the peripheral part of a sector defect might be discovered, the functional loss not being sufficient to produce a manifest alteration in response in the central part. This is an important reason for the use of more than one test-object and for the examination of a defect with minimal stimuli before forming a conclusion as to its limits.

(6) The *Margins* may be sloping, steep, or precipitous, according to the distance between the isopters at the edge of the defect, in other words, the transition between the most dense part of the defect and the seeing field may be gradual or abrupt. The gradient may vary at different parts of the edge of the defect and at different times in its progress.

Pathologically affected fields may be steep-edged as in retinitis pigmentosa, in which good central vision is retained in a very small field, or they may have sloping edges as in many cases of tabetic atrophy in which the depression of vision gradually increases towards the periphery.

(7) The expression "*Quality*" is intended, for want of a better term, to refer to certain features elicited by testing with red and blue and by examination in a dim light.

In the condition known as *torpor retinæ*, field changes which appear slight or indefinite with ordinary tests are intensified by reduction of the illumination. A characteristic feature is the disproportionate reduction in the perception of blue as compared with red, so that the recognition of blue, normally much more easy than that of red, may become more difficult. Depression is usually present at the same time, but may not be obvious, and the condition may manifest itself as a discrepancy between the fields found by the perimeter and those found by the screen in ordinary daylight. Thus the fields for white $\frac{1}{330}$, or blue $\frac{10}{330}$ may be practically normal, while those for white $\frac{3}{1000}$ or blue $\frac{60}{2000}$ may be greatly reduced without a proportionate reduction in the case of red. There is also reduction in the power of dark adaptation. This type of change is present in interference with the retinal outer layers, in detachment of the retina for example.

(8) *The Behaviour or Course*.—The onset of a field defect may be sudden, rapid, or gradual. It may remain stationary, progress, alter or fluctuate in respect of any of its characters. Both during the development and the retrogression of a defect, characters previously not evident may become manifest, and the observation by repeated perimetry of such changes may be of diagnostic importance. The behaviour of field changes is correlated rather to the nature than to the site of the causal condition.

II. Scotomata

A *scotoma* is an area of depressed vision within the field margin surrounded by an area of less depressed or of normal vision. It, therefore, corresponds to a lake or hollow on a hill, and is surrounded by isopters which are isolated from and not continuous with the more peripheral isopters. It is, however, not always convenient to adhere to the strict definition, and the expressions *scotoma* or *scotomatous area* are often used for any defect which extends deeply into the field without regard to the exact relations of the isopters concerned.

The periphery of the field may be intact or slightly depressed in the neighbourhood of the scotoma. A depressed area or channel of varying width and intensity may connect the scotoma with the periphery or with the blind spot or with both. This may be compared with a lake or tarn draining into the sea. This extension of a scotoma to the margin of the field is referred to as "breaking through." The characters of scotomata are the same as those of other field defects, but certain features require special consideration.

(1) *Position*.—A scotoma is said to be central when it involves the fixation area, paracentral when adjacent to it, and peripheral when it is outside the central area of the field. The terms central and paracentral are frequently used with a somewhat indefinite significance and the importance of scotomata of the central area renders a more exact terminology desirable. We distinguish, therefore, the *pericentral type*, in which the fixation area is more or less equally surrounded by the defect, as the *true central scotoma* and reserve the term *paracentral* for scotomata which approach or overlap the fixation area although mainly situated to one side. According to *their* relation to the fixation area such scotomata may be called *supracentral*, *infracentral*, *nasocentral* and *temporocentral*. In relation to the blind spot or area *cæca* scotomata

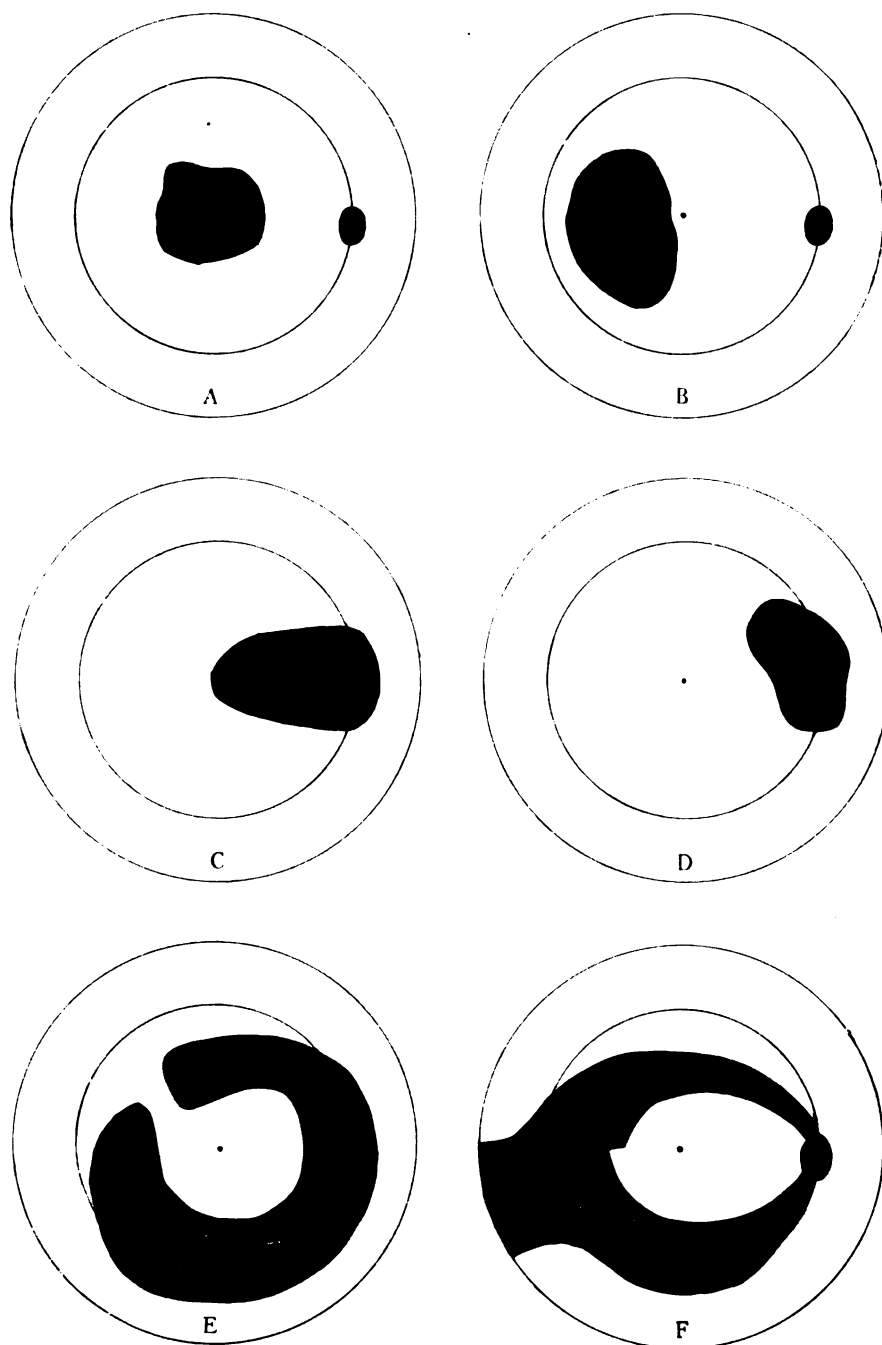


FIG. 25.—DIAGRAMMATIC REPRESENTATION OF DIFFERENT FORMS OF SCOTOMATA.

A. Pericentral scotoma. B. Paracentral scotoma. C. Centrocaecal scotoma with nucleus. D. Pericaecal scotoma. E. Annular scotoma. F. Double arcuate scotoma, nerve fibre bundle type, breaking through nasally and showing nasal step.

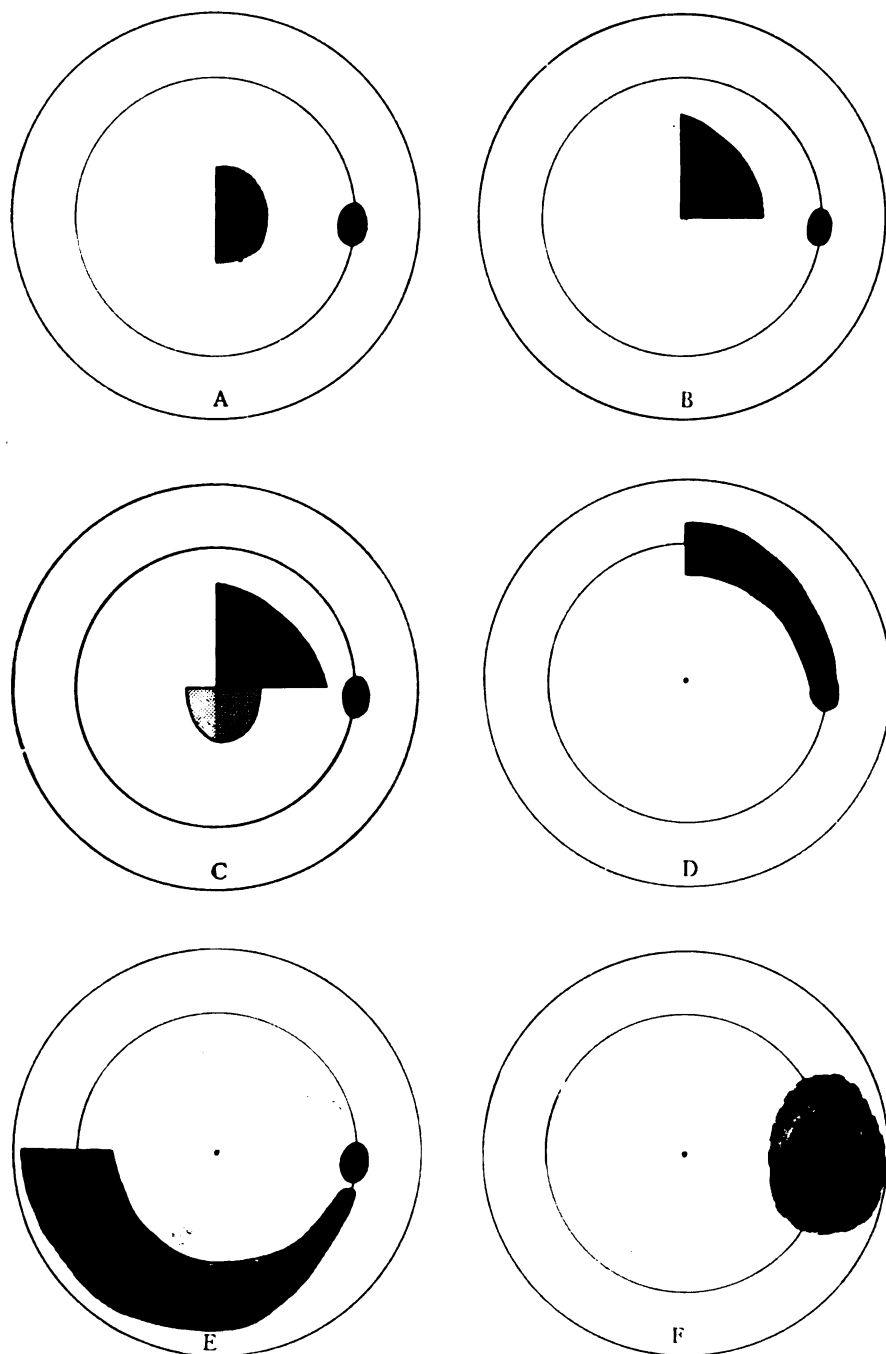


FIG. 26.—DIAGRAMMATIC REPRESENTATION OF DIFFERENT FORMS OF SCOTOMATA.

- A. Hemianopic central scotoma. B. Hemianopic quadrant central scotoma. C. Hemianopic quadrant central scotoma affecting several quadrants with varying intensity. D. Nerve fibre bundle scotoma, hemianopic type. E. Nerve fibre bundle scotoma, glaucoma type showing variation in intensity and nasal step. F. Enlargement of the blind spot.

are pericæcal when surrounding it and juxta- or paracæcal when adjoining it, the latter including supra-, infra-, naso-, and temporocæcal forms. Lastly, the common and important form which lies between and includes or extends to both fixation area and blind spot, corresponding in position to the papillo-macular area of the retina, is a centrocæcal scotoma. The expressions "enlargement" or "extension" of the blind spot are inexact as the so-called enlargement differs in several ways other than size from the physiological scotoma. Moreover, such terms suggest that the "enlargement" arises by an outgrowth from or expansion of the blind spot which is rarely the case. The amblyopic zone of the blind spot is important in connection with the estimation of the relationship of a juxtacæcal scotoma to the blind spot. In order to demonstrate the continuity or discontinuity of such a scotoma with the blind spot it is necessary to examine the interval between the two with a test-object large enough to be seen in that area under normal conditions, that is to say larger than $\frac{2}{2000}$. On the other hand when the edge of a scotoma is close to the blind spot but more than one or two degrees removed from it, a very small test-object ($\frac{1}{2000}$) should be used in order to determine whether the edge of the scotoma extends far enough to meet with the amblyopic zone. Allowance must also be made for a certain margin of error in a subjective examination of this kind as it is difficult for some patients to give accurate responses in respect of a narrow interval between two areas of amblyopia.

(2) *Shape*.—The shape of a scotoma is determined by the way in which the nerve elements involved are arranged in the retina :—

1. Circular or irregular.
2. Oval. This is nearly always an irregular horizontal oval, the centrocæcal form. The outline of the centrocæcal scotoma is frequently indented by a col or neck near the blind spot (Figs. 91D, 110, 111).
3. Cuneate or arcuate scotomata of nerve fibre bundle type. This form follows the lines of the nerve fibres in the retina and is therefore narrow near the blind spot, towards which one end is directed, and wider towards the field periphery. Defects of this type in the temporal field must be cuneate and in the nasal field arcuate. The arcuate scotoma is a cuneate scotoma bent over the macular area and resembles a comet or a Turkish scimitar. The form known as Bjerrum's scotoma is the best example of this group. Two opposed arcuate scotomata, one in the lower and one in the upper field, if equal and symmetrical, may form a ring scotoma. In this case the blind spot is always on the ring or, if the ring is incomplete, in the line of the ring. (See p. 82.)
4. In the zonular or annular type of ring scotoma the curve of the scotoma, although its concavity always faces the fixation point, does not follow the lines of the nerve fibres, and when a complete ring is present it may lie closely around the fixation area or may encircle both blind spot and fixation area. Scotomata of zonular type may involve any part of the field. Multiple concentric ring scotomata have been described and are probably functional in nature.

5. **Hemianopic.** A hemianopic central scotoma is a central hemianopia. The central field in this respect resembles the whole field in miniature, the apices only of the quadrants affected being defective. A quadrant scotoma is one which occupies the apex of one field quadrant. The terminology of hemianopic scotomata is the same as that of hemianopic defects of the whole field; they may be homonymous, bitemporal, etc. Two or more quadrant scotomata may be merged into a single defect, and, nevertheless, remain individually distinguishable by other characters, especially intensity. Sparing of the fixation area is uncommon in central hemianopic scotomata. Congruous homonymous paracentral scotomata also occur. These belong to the homonymous hemianopic group but are not bounded by the vertical or horizontal meridians of the field. Peripheral congruous homonymous scotomata probably also occur, but have not been reported.

(3) *Size.*—The size of a scotoma varies from a small spot of less than a degree in diameter to an area equal to almost the whole field. Since the apparent size is influenced by the strength of the stimulus used, it is necessary when testing scotomata of low intensity to reduce the stimulus in order to estimate the extent of the defect correctly. The different forms of scotoma may vary in size but it is the type and not the size which is of significance in diagnosis; the area is of more importance in prognosis. Attempts at precise measurement of the size, for example, of the blind spot, are therefore of secondary interest. A scotoma may extend at one part as far as the outer limit of the field. This is called “breaking through” (Fig. 25F).

(4) *Intensity.*—The intensity of a scotoma may vary from complete blindness to the slightest reduction of visual acuteness below the level normal for the retinal area involved. If the scotoma is small it may be impossible to demonstrate these extreme conditions, but in most cases the use of a series of test-objects will indicate sufficiently closely the degree of visual impairment. Where the patient's symptoms suggest the presence of a scotoma and field testing fails to demonstrate it, it is necessary to employ every practical means of diminishing the stimulus by using Bjerrum's screen, small test-objects coloured as well as white, and reduced illumination.

(5) *Uniformity.*—The intensity is seldom uniform. As a rule a scotoma is denser in the centre and less dense towards the periphery so that the smaller the test-object used the larger the scotomatous area appears. A common feature is the presence of a definite “nucleus” or focus of intensity in or near the middle of the scotoma (Fig. 25c). There may be more than one nucleus and the intensity diminishes from the nucleus towards the edge of the scotoma. A quadrant or hemianopic scotoma may be uniform all over, or may diminish in intensity from its apex towards the periphery. A curved band of greater intensity, indicating a portion of an arcuate scotoma, may be present, or a scotoma may be composed of quadrants of different intensities.

The demonstration of these features of the composition of a scotoma, or analysis, is of essential importance. The position and shape of the nucleus and the relationship of the parts of a hemianopic scotoma frequently have a diagnostic significance.

(6) *Margins.*—The margins of a scotoma may be sloping or steep. Good examples

are the scotomata of tobacco amblyopia and of acute retrobulbar neuritis respectively. A steep-edged scotoma which invades the fixation area is not incompatible with good central vision by Snellen's types ; if the edge is sloping vision is usually hazy. In the first instance the wandering movements of fixation (p. 50), provide incomplete but clear images, in the second indistinct images are introduced which produce a confusing effect.

(7) *Quality*.—In commotio retinæ, papillœdema and other conditions in which there is œdema of the macular area, it is of interest to study the relative perception of red and blue in the scotomatous area. The features already referred to (p. 63) may be found.

(8) *Behaviour*.—Variations in the course of scotomata are more common and more active than in that of defects of the field as a whole. This feature is related to the more usual causes of scotomata such as disseminated sclerosis. A scotoma may change its shape, or position, enlarge, diminish, become a ring scotoma or alter in intensity within a few days. The terms "wandering" or "fleeting" are applied to such scotomata. Or, alternatively, the changes may occur very slowly, or the scotoma may merely gradually disappear without variation in character.

From this survey of the normal and pathological field five essential points emerge :—

1. The field of vision should be thought of as a hill whose surface sinks and rises, and not as an area whose extent contracts and expands.
2. The condition of the visual field, whether normal or abnormal, cannot be ascertained by the determination of one isopter, that is to say, by the use of one test-object.
3. Changes in the field of vision are to be regarded and measured as depressions, not as contractions.
4. In all perimetric work the size of each test-object and its distance from the eye should be carefully selected with a view to the requirements of the individual case and should be recorded.
5. Any estimate of the significance of a field defect should be based upon the examination and consideration of *all* its characters.

We must now endeavour to ascertain what significance is to be attributed to alterations in the visual field.

CHAPTER VI

INTERPRETATION OF CHANGES IN THE VISUAL FIELDS

PERIMETRIC defects form the subjective expression of the site, extent, intensity, and behaviour of pathological changes in any part of the visual nerve path, and it is the aim of the perimetrist, first to ascertain as exactly as possible the presence and nature of field alterations, and, secondly, to endeavour to learn from his investigation what it can tell him as to the point affected and the morbid process concerned. The site of the interference, as, for example, in distinguishing between changes of subchiasmal, chiasmal, or suprachiasmal origin, is estimated by anatomical interpretation, which is based upon the shape and position of the field defects, while some guidance may be obtained as to the kind of morbid process present by the study of their onset, course and intensity, that is to say, by pathological interpretation.

Since the anatomical interpretation of field changes depends on their close relationship to the structure of the visual nerve path from the retina to the occipital cortex it is essential for the perimetrist to have a thorough knowledge, so far as that is possible, of the anatomy of the visual nerve path in its widest sense, including the relations of the pathway as a whole to adjacent structures, the arrangement of the different groups of cells and fibres within the pathway, and the most minute details as to its vascular supply. In some respects the field of vision and the anatomy of the visual nerve path may be regarded as complementary to one another, and much of what is known about either is due to the study of the other.

Omitting discussion on doubtful points, of which there are many, let us now examine the main anatomical features of the visual mechanism which concern the clinical perimetrist.

Retina

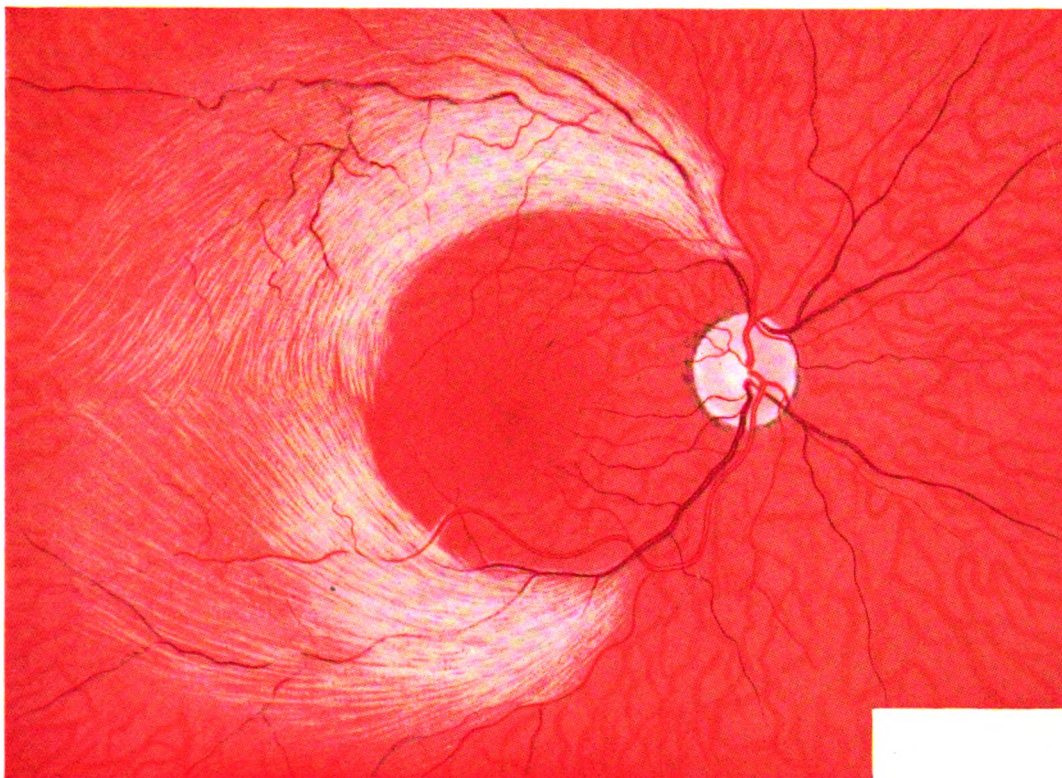
The retina may be regarded as consisting of two chief parts, the outer or photochemical stratum in which impulses are formed, and the inner or conducting stratum which conveys these impulses to the optic nerve. For our present purpose it is unnecessary to attempt to determine any sort of boundary at which the formation and elaboration of impulses cease and are replaced by conduction alone.

The important elements of the outer stratum are the rods and cones and the pigment epithelium. The fovea centralis contains only cones, the most central being very slender and concentrated, beyond the fovea the rods appear and gradually become more numerous until 4 or 5 mm. from the fovea three or four rods lie between every two cones.

Lauber (238) has suggested that the distribution of the rods and cones is connected with the relative positions of the isopters in the visual field. He found that anterior to the equator of the eye the cones are relatively more numerous on the nasal than on the temporal side.

Only at the optic nerve-head and the ora serrata is the retina firmly attached to the wall of the eyeball, elsewhere the rod and cone layer lies somewhat loosely on the

PLATE I.



OPAQUE NERVE FIBRES SHOWING THE ARRANGEMENT OF THE ARCULATE FIBRES IN THE RETINA, AND INDICATING THE POSITION OF THE HORIZONTAL RAPHE.

(With acknowledgements to Mr. Keith Lyle.)

[To face p. 70.]

pigment epithelium, which adheres to the choroid and separates from the rods and cones in retinal detachment. In detachment of the choroid or elevation of the retina by pathological changes in the choroid the pigment layer is not separated from the rest of the retina.

The conducting stratum contains the layer of ganglion cells and the nerve fibre or innermost layer. The ganglion cell layer is much thicker in the central area than elsewhere, and around the fovea the cells are heaped up, forming an elevated circular ridge higher on the nasal (papillary) side. The connections of these perifoveal cells with their cones slope inwards to the fovea, so that each ganglion cell lies at a little distance radially from its cone. Thus in the fovea itself there are no ganglion cells and the retina consists of pigment layer, cones, and a few cells of the inner nuclear layer.

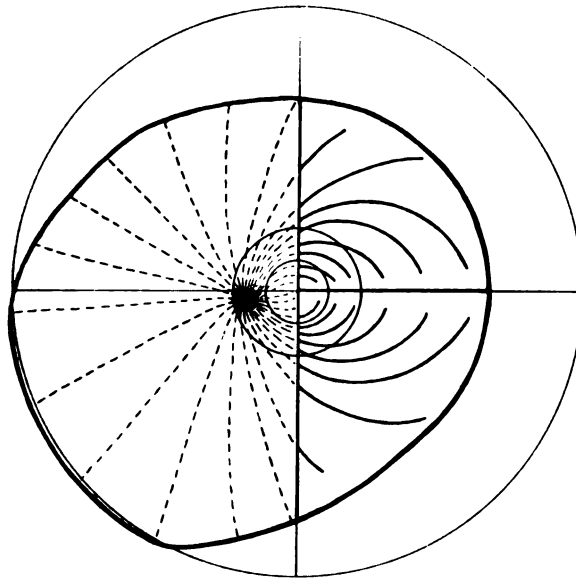


FIG. 27.—SCHEME OF THE PROJECTION OF RETINAL NERVE FIBRES IN THE FIELD OF THE LEFT EYE (AFTER ROENNE).

The horizontal raphé extends from the fixation point to the nasal margin. The continuous lines represent the uncrossed, the broken lines, the crossed fibres. The pericæcal and nearly the whole of the centrocæcal areas belong to the domain of the crossed fibres.

Around the exit of the optic nerve the outer retinal layers thin off somewhat and terminate with a sloping edge. The rod and cone layer extends farthest towards the nerve, but without reaching it, so that the area in which percipient elements are absent is larger than the area of the scleral foramen (Fig. 7). In front of this ring of modified retina lies the thickest part of the nerve fibre layer, thinning rapidly as the fibres spread outwards.

The blood vessels lie in the nerve fibre layer, and where the vascular trunks are large all the retinal layers are absent (see Appendix V.).

The arrangement of the fibres passing from the ganglion cells to the optic disc presents features of extreme importance for the perimetrist.

In that portion of the retina which is on the medial side of a vertical line passing

through the centre of the optic papilla, the nerve fibres are radially arranged, passing straight from the retinal periphery to the nasal side of the disc. Thus any group of fibres, which lie in juxtaposition at the nasal edge of the disc, regarded collectively, forms a cuneate or fan-shaped bundle. Such a bundle contains fibres from a sector of the retina, its widest part corresponds to an arc of the periphery, and its apex lies on the disc edge. Its size depends on the number of fibres included. Such a group is, therefore, not a true anatomical unit, as there is no segregation by septa or otherwise into groups of this kind, but pathological influences frequently pick out such bundles of fibres. They form very convenient and important descriptive units and are known as *nerve*

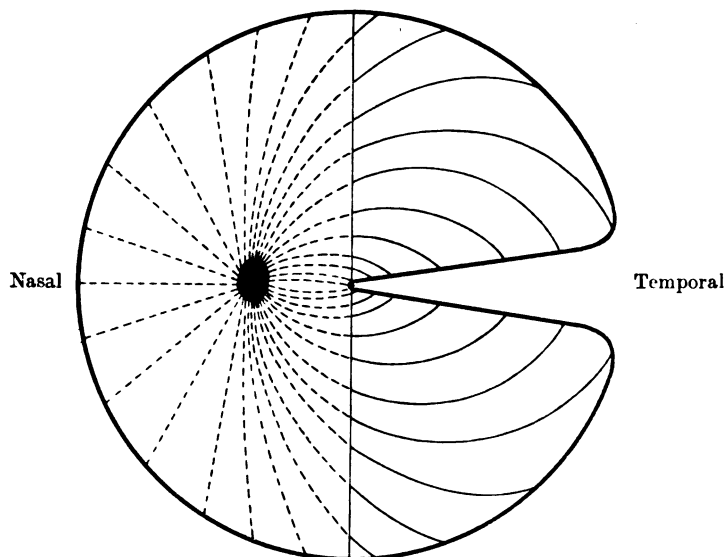


FIG. 28.—THE HORIZONTAL RAPHE.

Diagrammatic representation of the arrangement of the nerve fibres of the retina of the left eye seen from in front. The periphery of the retina is regarded as the edge from which the nerve fibres originate and upon which the most sensitive spot, the macula, is situated. At the lateral end of the horizontal meridian of the retina an infolding of the periphery with the macula at its apex is imagined. This fold progresses medially causing the uncrossed fibres passing from its upper and lower borders to become curved, in this way creating the arching fibres arising from the raphe, which is formed when the edges of the fold ultimately fall together. Thus the macula lies at the medial end of the raphe, which does not extend to the optic nerve, and the papillo-macular bundle comes to lie between the fibres from the upper and lower temporal retinal quadrants. The crossed fibres are indicated by broken lines, the uncrossed by continuous lines.

fibre bundles. (The crossed and uncrossed and the papillo-macular fasciculi are not included as nerve fibre bundles in this sense.)

On the temporal side of what we may call the papilla line the fibres traverse an arcuate course from the periphery to the disc curving over and under the central area, which is in this way encircled.

Extending from the fovea horizontally outwards to the temporal edge of the retina is an invisible line which is not crossed by any nerve fibres and which is known as the *horizontal raphe* of the retina. The nature of this raphe is most clearly grasped if we look upon it as a fold in the temporal margin of the retina drawn horizontally into the centre, the fovea being at its apex and its edges falling into apposition (Roenne 344). In this sense the raphe may be regarded as a part of the retinal periphery and the macula as

really situated upon the retinal edge. Those fibres which pass from the upper and lower parts of the temporal retina are only slightly curved ; as the horizontal meridian is approached the arching becomes more pronounced, and the fibres from the raphé and its neighbourhood traverse an almost semicircular course. The macular fibres are short and less curved, those lying near the horizontal meridian passing practically straight to the temporal side of the optic papilla. The nerve fibre bundles on the temporal side of the disc are therefore in all essentials similar to those on the nasal side ; they pass from the periphery, if we regard the raphé as a fold in the periphery, to the papilla ; they are fan-like or cuneate in shape ; but they are curved, whereas the nasal bundles are straight.

Thus the portion of the retina on the temporal side of a vertical line passing through the papilla contains arching fibres and is divided into upper and lower quadrants by the raphé and an imaginary line continuing it to the disc, while the portion on the nasal side of the papilla line contains radiating fibres and is undivided.

In addition to this local anatomical distribution of the nerve fibres there is a cerebral distribution to the two halves of the retina on each side of a vertical line passing through the fovea. The visual fibres from the temporal half-retina constitute the *uncrossed bundle* which passes to the tract and cortex of its own side. Those from the nasal half-retina form the *crossed bundle* which crosses at the chiasma to the visual centres of the opposite side. From this arrangement we see that the uncrossed bundle is represented in the retina entirely by the longer arching fibres from the temporal side of the vertical meridian, while the crossed bundle contains the straight radiating fibres from the retina on the nasal side of the papilla and also the arching fibres from the area between a vertical line through the fovea and another through the papilla. The retina around the papilla is entirely supplied by crossed fibres, and both crossed and uncrossed fibres enter into the composition of a complete arching bundle extending from the temporal periphery to the optic nerve. The peripheral part of such a bundle contains uncrossed fibres alone, but as soon as the vertical meridian is passed all the new fibres which are added are crossed.

The temporal half of one retina together with the nasal half of the other form the right or left homonymous half-retinæ as the case may be, and their visual fibres go to the same side of the brain. If the half-retinæ are coupled as binasal or bitemporal their nerve connections are with both sides of the brain through the crossed and uncrossed bundles respectively.

Optic Nerve-Head or Papilla

The nerve fibres are gathered together at the optic papilla to leave the eyeball as the optic nerve. At this level the nerve fibre layer attains its greatest thickness and is specially thick on the nasal side of the papilla owing to the accumulation of fibres, while the temporal side is left free for the papillo-macular bundle which displaces the temporal fibres to the upper and lower parts of the papilla, an arrangement which helps to make the physiological cup shallow or sloping temporally and deep or overhung at the upper, lower and nasal sides. At the upper and lower poles of the papilla the larger vascular trunks are grouped, at first intermingled with the nerve fibres, but soon becoming superficial. Individual variations are common.

Two views have been advanced as to the relationship between the peripheral and central retinal fibres as they pass into the papilla. According to some authorities the peripheral fibres pass to the centre and the peripapillary fibres to the periphery of the

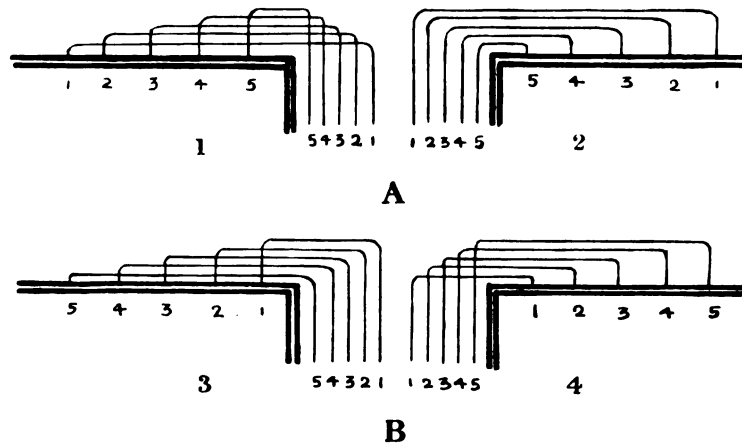


FIG. 29.—DIAGRAM SHOWING THE VARIOUS WAYS IN WHICH THE NERVE FIBRES MIGHT BE ARRANGED AS THEY ENTER THE OPTIC NERVE HEAD.

1 and 2. The fibres from the retinal periphery pass to the centre of the nerve and the peripapillary fibres to the periphery of the nerve.

3 and 4. The fibres from the retinal periphery pass to the periphery of the nerve and the peripapillary fibres to the centre of the nerve. (Modified from Van der Hoeve (441).)

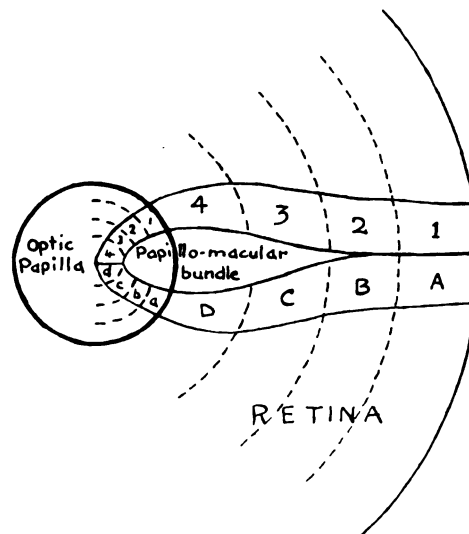
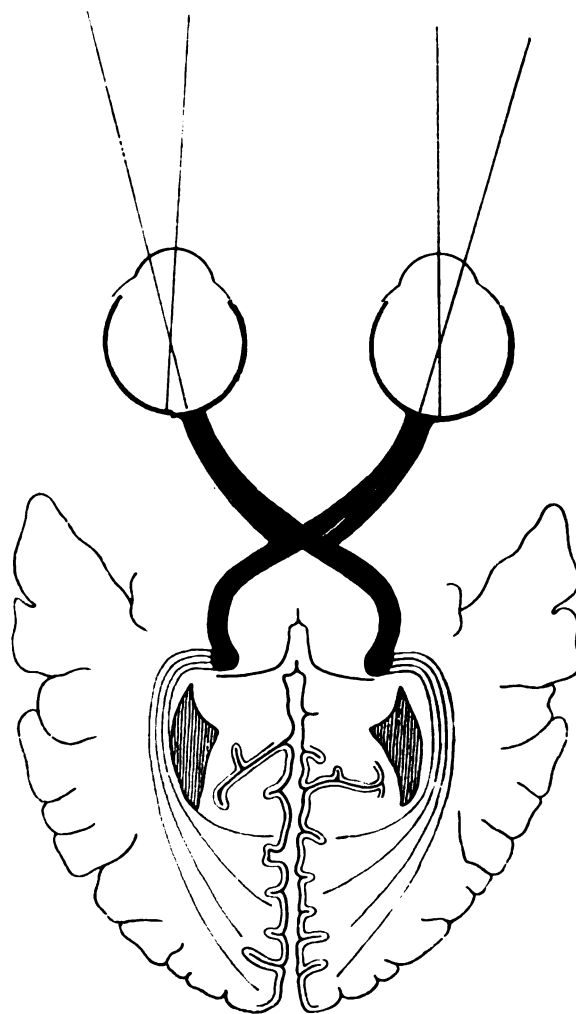


FIG. 30.—DIAGRAM INDICATING THE RELATIONSHIP OF THE PAPILLOMACULAR FIBRES TO THE PERIPHERAL RETINAL FIBRES AS THEY ENTER THE OPTIC PAPILLA. (AFTER WILBRAND AND SAENGER.)

nerve, at least in its anterior portion. Others believe that the fibres from the **retinal** periphery pass to the outside of the nerve and the peripapillary fibres to its centre (Von Hippel 444). The question is important in regard to the interpretation of field defects for, according to the first view, peripheral interference in the nerve, unless posteriorly

PLATE II.



THE VISUAL NERVE PATHS SHOWING LINES OF PROJECTION OF
THE FIXATION AREA AND THE BLIND SPOT.

[To face p. 74.]

PLATE III.

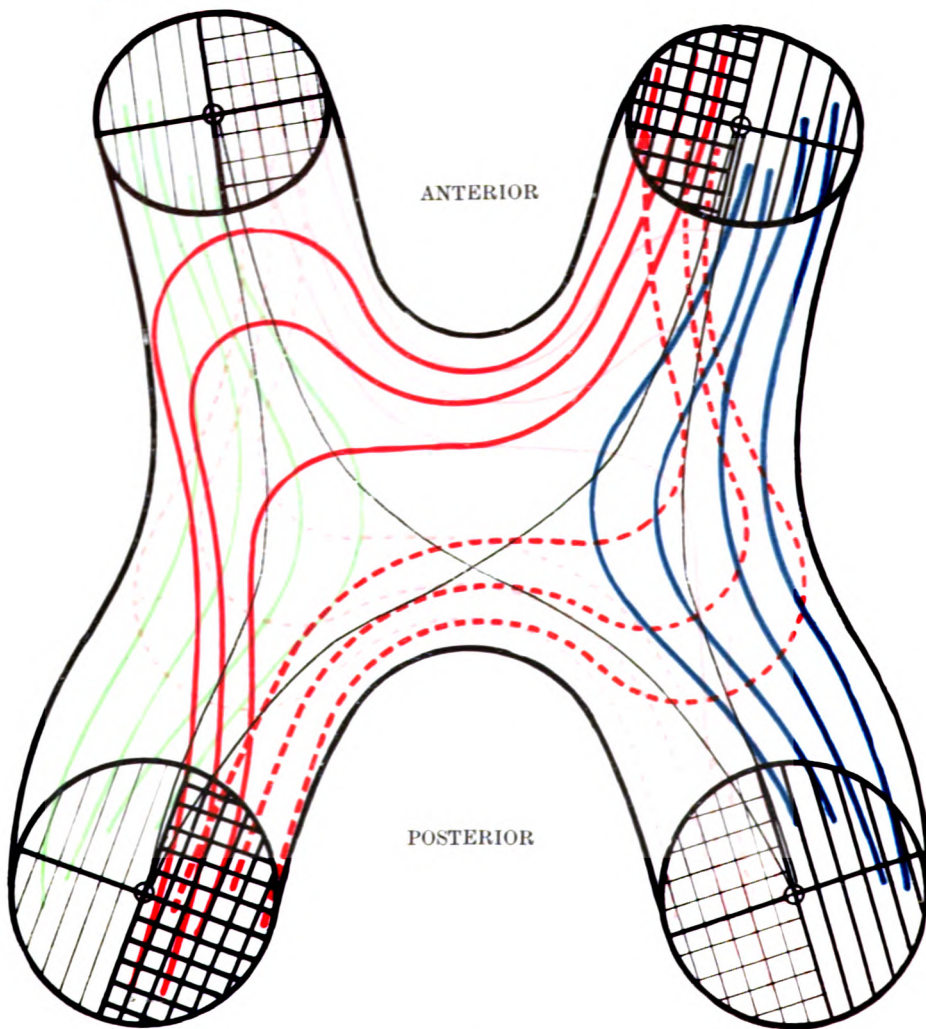


DIAGRAM TO SHOW THE PROBABLE ARRANGEMENT OF THE CHIASMAL CROSSING.

Continuous red lines indicate crossed fibres from the lower medial quadrants of the optic nerve ; broken red lines crossed fibres from the upper medial quadrants ; continuous blue lines uncrossed fibres from the lateral quadrants.

In order to avoid confusion the fibres from the right optic nerve are coloured brightly, those from the left nerve faintly.

The crossed fibres from the lower medial quadrant of the right optic nerve (continuous red line) are shown crossing in the anterior part of the chiasma and passing forwards into the termination of the left optic nerve, forming a knee there, and then backwards to the medial side of the left optic tract. The crossed fibres from the upper medial quadrant of the right optic nerve (broken red line) pass backwards along the same side of the chiasma to its posterior angle and then across its posterior part to the upper medial quadrant of the opposite tract.

The uncrossed fibres from the lateral half of the nerve are spread out in the body of the chiasma so that those from the upper quadrant lie medial to those from the lower.

At each side of the chiasma crossed fibres from both sides are mingled with uncrossed fibres of the same side. The position of the macular fibres is indicated by the thin black lines. These fibres occupy a relatively large area in the postero-inferior part of the chiasma and are therefore easily affected by interference from below and behind.

[To face p. 75.]

situated, would cause a pericæcal scotoma or "enlargement of the blind spot," while if the second be correct, peripheral restriction of the field would result. It is probable that the fibres are mingled in the retina and that their relationship alters as they enter the nerve, the peripapillary fibres being peripheral at the papilla, but becoming more central after the lamina cribrosa is passed. On this assumption a retinal sector or a nerve fibre bundle would occupy approximately a sector of the nerve-head, the apex of the nerve sector receiving the peripheral retinal fibres and the periphery of the nerve sector the peripapillary fibres. In such an arrangement the foveal fibres, being really peripheral fibres, would pass to the apex of the temporal quadrant, *i.e.* to the centre, of the nerve.

Histological examination has not yet shown more than an approximate topographical relationship between the retina and the optic nerve.

The anatomical features of the papilla probably exert an important influence in determining the exact way in which the field changes develop in individual cases of glaucoma. Their relation to the normal blind spot has already been referred to.

Optic Nerve

The course and relationship of the fibres from the different retinal quadrants after they leave the eyeball are not yet definitely known in full detail. In general arrangement a sector of the retina is approximately represented by the corresponding sector of the nerve, and the superior, inferior, lateral and medial fibres retain their respective positions throughout the whole oculo-occipital visual path. For the more exact architecture of the visual path we may accept for the present the main features of Henschen's (165) well-known description (Fig. 31), although there are difficulties in regard to details.

Immediately behind the eye the fibres from the nasal half of the retina, the crossed bundle, occupy the medial quadrant of the nerve, those from the central area, the papillo-macular bundle, the lateral quadrant. The crossed fibres occupy about three-fifths of the nerve section. The fibres from the temporal retina, the uncrossed bundle, are separated by the papillo-macular bundle into superior and inferior groups which pass into the superior and inferior quadrants of the nerve. This grouping would necessitate a re-arrangement between the retina and the beginning of the nerve since, as we have seen, both crossed and uncrossed fibres pass the edge of the papilla together in the same bundle. This is evident from the ophthalmoscopic study of opaque nerve fibres and from the arcuate scotomata of glaucoma and other conditions. The part of the retina which lies between a vertical line through the macula and a similar line through the papilla gives rise to crossed fibres which, excepting those belonging to the papillo-macular bundle, pass into the upper and lower poles of the papilla together with the uncrossed fibres from the superior and inferior temporal retinal quadrants. If the bundles are already segregated immediately behind the eyeball the crossed elements must leave the uncrossed in the neighbourhood of the lamina cribrosa.

More posteriorly the two portions of the uncrossed bundle come together so that about the level of the entrance of the central retinal artery the macular fibres form a central core, the medial and lateral sides of the nerve being occupied by the crossed and uncrossed fibres respectively. At this part a cross-section of the nerve corresponds

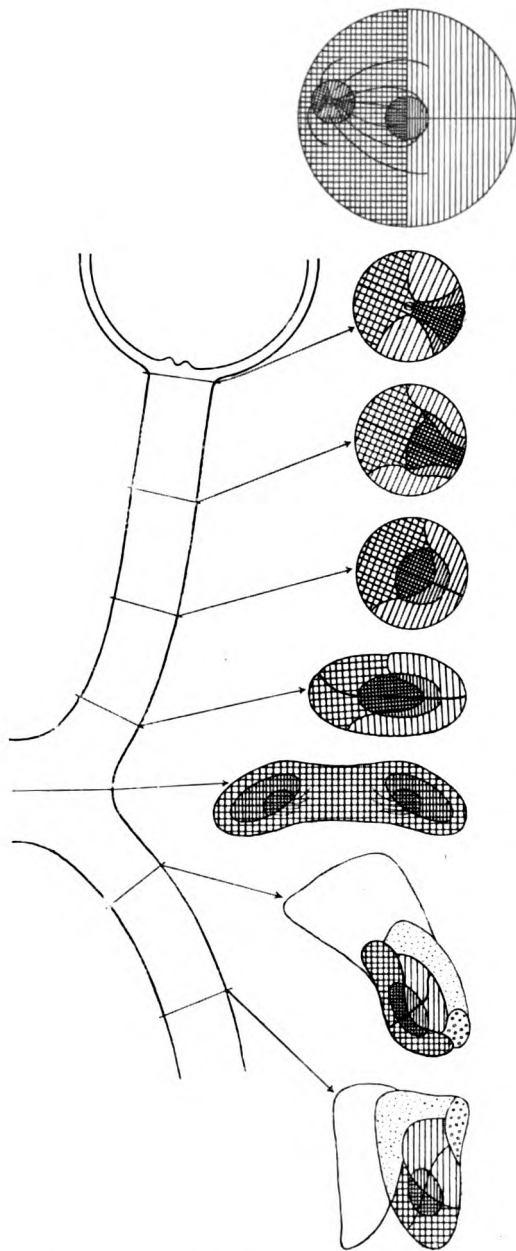


FIG. 31.—HENSCHEN'S SCHEME (MODIFIED) OF THE DISTRIBUTION AND COURSE OF THE NERVE FIBRES IN THE RETINA, OPTIC NERVE, AND TRACT (RIGHT SIDE).

The chiasmatal section is slightly modified from Wilbrand and Saenger.

approximately to the retina and a sector or zone in the nerve contains the fibres from a similar retinal area.

In the terminal part of the nerve immediately in front of the chiasma a strong glial septum enters its upper surface and extends obliquely downwards and medially in a slanting direction splitting off, as it were, a supero-medial mass of fibres (Fig. 256). These are the crossed fibres and in this way they become separated from the uncrossed, so that a lesion about this level may affect either predominantly or solely. The crossed fasciculus occupies the medial and infero-medial part of the nerve, the direct fibres the supero-lateral and lateral part.

At this level the nerve also contains, in its lower part, the knee of crossed fibres from the opposite nerve (Plate III).

Chiasma

In the chiasma the fibres of both nerves spread out and intermingle. The crossed bundle assumes a roughly quadrilateral outline extending to the margins of the chiasma before it is collected again to the medial part of the opposite tract. Although much labour has been expended on the problem of the chiasmatal crossing the arrangement of the fibres is still somewhat uncertain, and the following description, based on Wilbrand and Saenger, may be modified in the future (Plate III). Those fibres which come from the inferior part of the crossed bundle in the nerve, originally from the lower medial retinal quadrant, cross in the lower part of the front of the chiasma. The more anteriorly placed members of this group, after crossing turn forwards into the termination of the opposite optic nerve, where they form a loop or knee in its under-surface and then turn backwards along the side of the chiasma to the beginning of the tract, in which they occupy the infero-medial quadrant. The upper fibres of the crossed bundle in the nerve, coming from the upper medial retinal quadrant, cross in the middle and posterior parts of the chiasma near its lower surface and pass into

the supero-medial quadrant of the opposite tract. The more posterior fibres of this group pass backwards along their own side of the chiasma before they cross, and enter the commencement of the homolateral tract where they form a loop similar to but blunter than that formed by the anterior crossing fibres in the optic nerve. They then cross at the posterior border of the chiasma.

The uncrossed bundle passes from the upper lateral part of the nerve along the same side of the chiasma to the upper lateral part of the tract. As it passes backwards it forms a somewhat flattened oval band in the lateral part of each half of the chiasma. At first it lies at the edge of the chiasma sparsely interwoven with crossed fibres which farther backwards closely mix with and surround it. Clinical evidence suggests that the upper uncrossed fibres from the upper lateral retinal quadrant lie on the medial side of the uncrossed bundle in the chiasma and extend farthest towards the middle of the chiasma, and that the lower fibres, from the lower lateral retinal quadrant, lie more laterally along the side of the chiasma. At any rate, in median chiasmal interference from below, as in hypophysial tumours, the lower uncrossed fibres are the last to be involved.

Optic Tract

At the commencement of the tract the crossed and uncrossed bundles approach each other from the chiasma, the uncrossed fibres being supero-lateral and the crossed infero-medial. The fibres preserve their relative positions throughout with little change before entering the lateral geniculate body. According to Minkowski the fascicular segregation in the tract is not well defined. He distinguishes an infero-medial crossed bundle and a supero-lateral uncrossed bundle united by a middle mixed area. Le Gros Clark found that the crossed and uncrossed fibres are evenly intermingled in the terminal part of the tract but become separated in the geniculate body to reach their appropriate laminae. Clinical evidence, however, shows that the crossed and uncrossed bundles are sufficiently separate, at least in the lower part of the tract, to allow of the production by a lesion of a defect of one field only or of homonymous hemianopic defects unequal in the two fields.

Lateral Geniculate Body

The visual fibres in the optic tract pass to the lateral geniculate body. According to Henschen the relative positions of the different fibre groups are maintained at this level. The cells in the lateral geniculate body, in which the visual fibres from the tract terminate, are arranged in laminae to which the crossed and uncrossed fibres are separately distributed. The nasal periphery of the opposite retina is probably represented in the anterior third of the nucleus and the more central parts in regular order towards its posterior end, the macula in the intermediate part of the posterior two thirds.

Putnam and Le Gros Clark found that the homonymous upper retinal quadrants are projected on the medial half of the nucleus and the corresponding lower retinal quadrants on the lateral half.

Geniculo-Calcarine Path

From the lateral geniculate body the visual fibres pass into the retrolenticular portion of the posterior limb of the internal capsule, whence they emerge as the optic

radiation or geniculo-calcarine path terminating in the *area striata* of the occipital lobe. In the internal capsule they lie behind the sensory fibres and medial to the auditory bundle. The fibres from the upper retinal quadrants lie superiorly, those from the lower retinal quadrants inferiorly, in the radiation and are anatomically segregated by the interposition of the macular fibres.

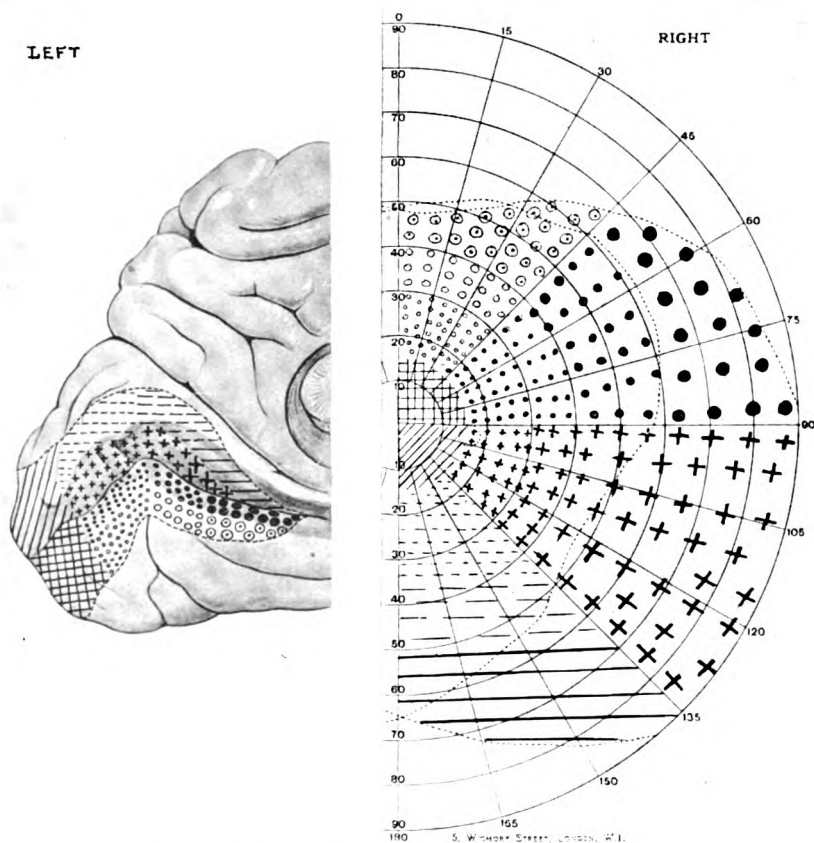


FIG. 32.—THE CORTICAL RETINA (GORDON HOLMES).

Diagrammatic representation of the cortical retina. The calcarine and post-calcarine sulci are represented as continuous and widely opened. The macular area is relatively large, the peripheral area relatively small, and the visual centre extends farther forwards on the lower lip of the calcarine sulcus than on the upper.

Cortical Visual Centre

The cortical visual centre occupies the *area striata* on the medial surface of the occipital lobe extending forwards from the occipital pole on both lips of the post-calcarine sulcus and on the lower lip only of the calcarine sulcus. At the occipital pole it is found for a short distance on the lateral aspect of the occipital lobe.

Each centre corresponds to the two homonymous half-retinæ of its own side and may be regarded as a cortical retina representing the ocular retina from periphery to centre from before backwards. Its most anterior part represents the nasal periphery of the opposite retina, which corresponds to the unpaired temporal part of the binocular

visual field, and receives only crossed fibres. Behind this the concentric zones of the two homonymous half-retinæ are represented in succession, the most posterior part corresponding to the macula. The upper and lower retinal areas correspond respectively to those parts of the cortex which lie above and below the post-calcarine sulcus, the bottom of which represents the horizontal meridian of the retina. The medial periphery of the retina, which corresponds to the temporal crescent in the field, is probably represented entirely in the lower lip of the calcarine sulcus.

For further notes on the anatomy of the visual path and cortex the reader is referred to the Appendix III, p. 291.

Macular Fibres

The macular fibres come from the apical or central portions of the four retinal quadrants and are collected together to a sector shaped area occupying about a third of the papilla at its lateral side. In the retina they lie between the fibres from the upper and lower quadrants and this arrangement is continued up to the occipital cortex. According to Henschen's description, immediately behind the eye the central part of this sector contains the superior and inferior crossed macular fibres, while its upper and lower parts, which lie adjacent to the superior and inferior uncrossed peripheral bundles, contain the superior and inferior uncrossed macular bundles, which are thus separated from each other. The macular fibres gradually move towards the centre of the nerve and form a central bundle consisting of uncrossed fibres laterally and crossed fibres medially, enveloped by the peripheral fibres which are similarly arranged. In the posterior part of the orbital portion of the nerve the macular fibres form an axial core (Fig. 31). Immediately in front of the chiasma the crossed macular fibres, as Roenne (324) has shown, separate from the uncrossed and turn upwards to the infero-lateral side of the glial septum which enters the nerve obliquely downwards and inwards at this part. They lie superiorly in the chiasma and cross in the upper layers of its middle and posterior thirds. The posterior edge of the chiasma consists entirely of crossing central fibres, an anatomical feature which is probably related to the frequent appearance of central hemianopic scotomata in chiasmal pressure interference from below. The uncrossed macular fibres are infero-central in the termination of the optic nerve and lie centrally in each lateral half of the chiasma and centrally in the tract.

On clinical grounds it seems probable that the macular fibres follow the arrangement of those from the periphery and form, as it were, a little chiasma within the chiasma and towards its posterior part.

In the tract Henschen's scheme shows the macular bundle central in position, the relative arrangement of the fasciculi following that of the peripheral fibres. According to Roenne the crossed macular fibres enter the tract above the uncrossed. Throughout its course the macular bundle lies closely along the connection of the tract to the brain, so that it is found at first in the central and supero-medial part of the tract, and, as the latter rotates outwards on its axis, becomes supero-central and finally supero-lateral and peripheral, in which position it enters the geniculate body.

The fibres then pass to the superior layer of the middle and posterior parts of the

nucleus and to the major part of its posterior extremity. According to Malbran (277) they terminate in the anterior part of the geniculate body.

In the radiation the macular fibres form a segregated bundle separating the upper from the lower peripheral fibres. They terminate in the most posterior part of the occipital centre and occupy a relatively large share of the visual cortex. The foveal fibres are represented at the tip of the occipital pole.

Although some observers have strongly advocated the view that both sides of each macular area are represented in each cortical macular centre, it seems practically certain that this is not the case and that each cortical macular centre represents only the homolateral halves of the two maculæ.

Several points require further consideration. We have seen that both crossed and uncrossed fibres may lie together in the same arcuate bundle in the retina and at least as far as the papilla. There is clinical evidence to show that a lesion in the nerve trunk may also involve fibres which in the retina form an arching bundle containing both crossed and uncrossed fibres. If the segregation of the fibres is as described such a lesion must involve the boundary between the crossed and uncrossed fasciculi. It is apparent that fibres which lie together in the retina continue to lie together in the nerve, and that a fibre group which can be interrupted by a localised lesion at the papilla edge is so shaped and placed in the nerve trunk as to be capable of isolated or almost isolated interruption there also. The fibres remain in such groups at least as far as the chiasma, where the crossed and uncrossed fibres become separated, so that the groups now contain only one kind of fibre, and a group or bundle lesion at this level produces a defect bounded by the vertical meridian of the field.

The microscopic examination of degenerations following experimental retinal wounds has frequently shown diffuse rather than segregated changes in the nerve. It is possible, however, that some of the degenerated fibres seen are not visual fibres. Henschen's scheme cannot be regarded as final, and further research is required.

To sum up: it may be regarded as established that there is a segregation of the fibres in the visual path into fasciculi approximately according to the arrangement indicated, though the demarcation between the different groups is probably not very exact and individual variations may occur.

We have seen that the optic nerve corresponds to the retina and that the optic tract corresponds to the homolateral half of the combined retina. At some point above the commencement of the tract the crossed and uncrossed fibres become intimately mixed in such a way that fibres from corresponding parts of the two retina lie together. Above this level each fibre group corresponds to an area of the binocular retina and a minute lesion produces homonymous field defects. According to Roenne, the crossed fibres place themselves side by side with their uncrossed fellows in the lower part of the tract; according to Henschen, the juxtaposition takes place in the geniculate body. Minkowski believed that the crossed and uncrossed fibres are still separate in the geniculate body and that the intermixture is closest in the radiations and less so in the cortex. The clinical evidence favours Roenne's view.

An exception occurs in the case of the fibres which come from the medial periphery of the retina. As there is no corresponding temporal retinal area this group of crossed fibres has no uncrossed companions. Whether these fibres are segregated throughout the visual path is uncertain, but the clinical evidence indicates that in the geniculocalcarine portion they lie on the medial side of the optic radiation and terminate in the most anterior part of the visual cortex.* A lesion here will, therefore, affect only the unpaired portion of the opposite temporal field producing a crescentic peripheral restriction, or, on the other hand, a lesion may affect the other elements of the path and spare this group, giving rise to a homonymous hemianopia with a retained crescentic area at the periphery of the affected temporal field (Figs. 227, 238).

Further notes on the anatomy of the visual path will be found in the Appendix.

ANATOMICAL INTERPRETATION

The anatomical interpretation of field defects is the correlation of their position, shape and extent, and sometimes of their quality, with the anatomy of the nerve path, in order to determine as far as possible the *site* of the lesion.

Retina

Defects due to retinal lesions depend on involvement of the percipient cells, the outer stratum or photochemical apparatus, or of the ganglion cells and nerve fibres, the inner stratum or conducting apparatus.

When the percipient elements or ganglion cells are affected, without involvement of the nerve fibre layer, the field defect corresponds to the retinal lesion in position, shape, extent and intensity. Such defects are usually more or less round, oval or irregular in shape. A relative reduction in the perception of blue as compared with that of red (acquired tritanopia), indicates a lesion of the photochemical apparatus. In conduction defects, even of low intensity, the superiority of vision for blue over that for red is usually very pronounced. In practice this distinction may not be easy to establish, for, when the impairment of the percipient elements is severe, vision for red is also reduced (see Chapter VII).

Exaggeration of the defect in dim light is also characteristic of outer layer lesions. These tests are of value in certain cases, especially when other indications suggest outer stratum involvement and ophthalmoscopic examination is difficult or inconclusive.

The scotoma in retinal disease frequently assumes an annular form as in retinitis pigmentosa and other conditions. The ring may be very small and immediately pericentral, or may be more peripheral and larger, or may be incomplete. The zonular shape has not yet been satisfactorily explained; in some cases it may be due to the great superiority of central vision over that of the immediately surrounding elements so that a certain degree of visual depression may be sufficient to become manifest in the adjoining pericentral area though not at the centre itself. These defects should not be confused with nerve fibre bundle arcuate scotomata, which they may resemble when they are so situated as to correspond to the lines of the arching fibre bundles.

* See Appendix III., p. 291.

Defects due to damage to the ganglion cells present the features of impaired conduction—relative red-green blindness and reduced perception of white. The field changes correspond to the projection of the affected area of the retina and present, as far as is known, no features which identify them with ganglion cell lesions. They resemble many scotomata due to lesions of the optic nerve, though they differ markedly from defects due to interruption of the nerve fibres in the retina, since nerve fibre bundle defects are never present. The usual forms are centrocæcal or approximately round or irregular central scotomata, or peripheral restriction with or without wide sector defects.

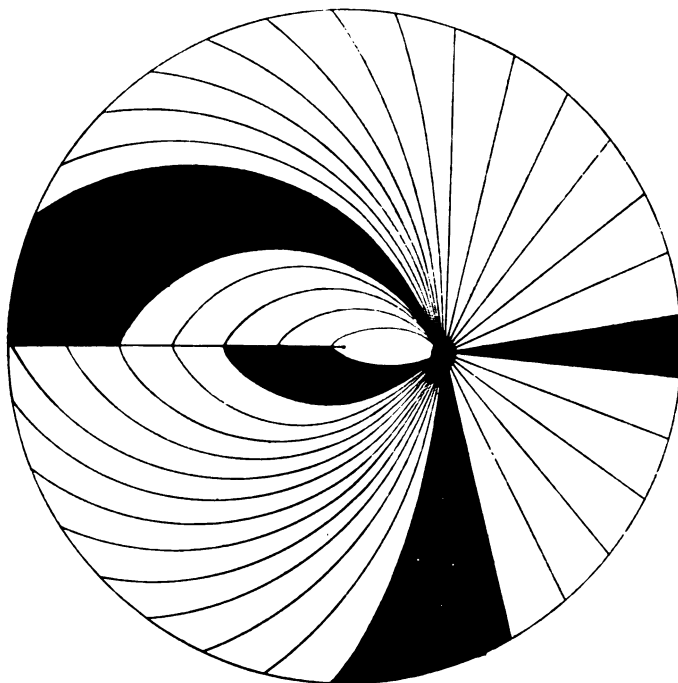


FIG. 33.—DIAGRAMMATIC REPRESENTATION OF THE PROJECTION OF THE NERVE FIBRES OF THE RIGHT RETINA, SHOWING FOUR TYPICAL NERVE FIBRE BUNDLE DEFECTS. THE ARCUATE SCOTOMATA SHOW NASAL STEPS.

Lesions of the nerve-fibre layer produce defects which correspond not to the area of the lesion, but to the area from which the affected fibres come. A small lesion, if near the disc, may therefore produce a very extensive field defect. The intensity of the defect reflects the degree of impairment of conduction, that is, the severity of the lesion.

It is evident from anatomical considerations that a small circumscribed lesion involving the whole thickness of the nerve fibre layer near the optic disc on the nasal side will produce a fan-shaped or cuneate defect in the temporal field with its apex towards or joining the blind spot and its broad end at the temporal periphery, while a lesion at the upper or lower end of the disc will

produce an arcuate or comet-like scotoma curving from the blind spot respectively under or over the central area and ending on the horizontal raphe or elsewhere on the nasal periphery.

The depth of the lesion is approximately indicated by the extent of the defect according to the distribution of the fibres involved.

Such scotomata are known as nerve fibre bundle defects because they reflect the retinal distribution of the nerve fibres as they converge towards the papilla.

Strictly speaking, every field defect, which is not caused by damage to cells, is due to a fibre lesion, and, when adjacent fibres are involved, to a bundle lesion. The term is, however, widely accepted as a convenient expression to indicate a defect which corresponds to a group of fibres which lie together in the retina. Such defects, both arcuate

and cuneate—for the arcuate defect is merely a cuneate defect bent over the fixation area, just as the arcuate bundle is a cuneate bundle bent over the macula—are not produced by retinal lesions only, but also by lesions at higher levels. The nerve fibre bundle defect is, therefore, not a distinctive sign of a retinal lesion. An arcuate defect depending on a lesion in the nerve may cross the vertical meridian of the field showing that both crossed and uncrossed fibres are involved, and, therefore, that fibres which lie together in an arcuate bundle in the retina continue to lie together in the nerve, at any rate, in such relationship that they can be closely picked out by the morbid process concerned. Arcuate scotomata due to chiasmal lesions are hemianopic in type, being bounded by the vertical meridian of the field, showing that the retinal arrangement of the fibres persists at this level though modified by the separation of the crossed from the uncrossed fibres. At higher levels the production of nerve fibre defects is uncertain.

The most typical field changes of this type are those caused by a lesion of the fibre layer of the retina at or near the edge of the optic disc as in glaucoma. One end of the defect is connected to the blind spot at either its upper or lower margin, or, in incomplete instances, is so directed that, if continued, it would join the blind spot. The other end lies along the horizontal meridian in the nasal half of the field or extends to the nasal periphery; the intermediate part arches around the fixation point in a larger or smaller curve widening as it leaves the blind spot so that the scotoma is somewhat comet-shaped. There may be a curved scotoma in contact with the upper or lower end of the blind spot and extending for a variable distance or it may be isolated both from the blind spot and the horizontal meridian. Such defects, whether joined to the blind spot or not, are often scimitar-shaped or crescentic, and if double and symmetrical (*i.e.*, one above and one below the horizontal meridian) may form a ring scotoma, the blind spot representing the stone set in the ring. The extent and intensity of scotomata of this kind correspond to the amount of interference at the disc margin; if the obstruction is complete the defect extends from the blind spot to the horizontal meridian, and, if a long and wide bundle is affected, to the nasal periphery of the field. The straight edge along the horizontal meridian constitutes Roenne's *nasal step*, which is most clearly defined when one arcuate scotoma alone, or two opposed arcuate scotomata of unequal size, are present. The horizontal meridian will then be bared along one side only or to unequal extents on both sides. It is the projecting bared portion of the horizontal meridian which forms the step; if two opposed arcuate scotomata of equal size are present there is no step since there is no projecting bared part of the meridian. Between the fixation point and the blind spot the edge of the defect is curved, not straight, as the retinal raphé extends only to the fovea. These scotomata may vary in intensity in different parts, which explains to some extent the different forms which have been recorded, for it may happen that only the more intense portion is discovered. In some cases a small visual angle is required to expose the whole extent of the defect. If the injured nerve fibre bundle is on the nasal side of the optic disc the resulting field defect is wedge-shaped, with its apex at the blind spot or directed towards it if the bundle is only partially interrupted. Thus nerve fibre bundle scotomata, due to papillary or juxta-papillary lesions, reflect the arrangement of the nerve fibres in the retina, and are

all cuneate defects radiating from the blind spot to the periphery. The arcuate type is a cuneate defect bent over the fixation point and terminating upon a horizontal line, which we may regard as in a sense a part of the periphery, since it corresponds to the horizontal raphé which, as already pointed out, may be looked upon as a fold in the retinal margin (Fig. 28).

Optic Nerve

Since the cross-section of the nerve corresponds in its orientation with the retina, the position of a lesion of the nerve trunk is reflected inversely in the field. Interference with one side of the nerve is associated with a defect in the opposite side of the field, while peripheral and central impairment are manifested by peripheral and central visual loss respectively. A sector in the field corresponds to the opposite sector in the nerve and ring scotomata, whether of zonular or of nerve fibre bundle type, are probably due to zonular interference in the nerve. Defects of hemianopic appearance due to a lesion at this level are never sharply demarcated along the vertical meridian, and if thoroughly examined, often show considerable irregularity.

This interpretation holds good for the major portion of the nerve trunk, but at its beginning and termination the arrangement of the fibres introduces certain modifications. A lesion in the periphery of the nerve immediately behind the eyeball would involve the macular fibres owing to their position at this level, and probably certain cases of combined central and peripheral field defect are due to interference in this situation. At the chiasmal termination of the nerve the diversion of the crossed from the uncrossed fibres permits of the predominant involvement of one of these groups, more especially as regards the macular fibres, and the production of uniocular hemianopic defects. Scotomata of this type may be called "junction scotomata" on account of their site of origin at the junction of the nerve and the chiasma. According to Wilbrand and Saenger, the involvement of the knee of crossed fibres from the opposite nerve may be the source of a peripheral upper temporal defect in the opposite field, so that a lesion of one optic nerve at its junction with the chiasma may produce defects in both fields.

Finally, it must be remembered that lesions are longitudinal as well as transverse, and different parts of the nerve may be involved at different levels by the same lesion.

It is evident that subchiasmal lesions can be distinguished from those of chiasmal or suprachiasmal situation by the absence of hemianopic characters, that is, of features which indicate the special involvement of the crossed or uncrossed fibres of both eyes. A uniocular field defect which extends to both sides of the vertical meridian must be of subchiasmal origin. Bilateral field defects of this type must be due to bilateral nerve lesions since, if due to chiasmal interference, the lesion would require to be so placed and of such intensity throughout as to produce an equal degree of impairment of the different fibre bundles. The anatomical arrangement of the chiasma prevents this, and bilateral defects which occupy both sides of the vertical meridian of the field can always be shown by quantitative perimetric analysis to be of hemianopic type if they result from lesions situated at or above the chiasma.

In the subchiasmal path, however, the exact site of a lesion is betrayed only to a limited extent by the shape and position of the field changes produced. Depressions

of various kinds and arcuate and ring scotomata, similar in form, may be produced by retinal as well as nerve lesions, and, with the exception of some unocular defects of hemianopic type, the field changes do not indicate the level in the nerve trunk at which the lesion is situated. The centrocæcal scotoma, for example, so common as a result of subchiasmal interference, does not appear to be related to any special situation.

Pericæcal or juxtacæcal scotoma (enlargement of the blind spot) may be due to a lesion of the retinal cells or fibres surrounding the optic papilla, or to a lesion of the fibres in the nerve which come from the peripapillary part of the retina. The diagnostic significance of changes in the size of the blind spot depends not on the amount of the enlargement but, as in the case of any other scotoma, on the type of alteration.

Chiasma

In the chiasma the arrangement of the nerve fibres causes lesions to produce defects of bitemporal hemianopic type. Variations in the position of the lesion cause differences in detail but do not alter the type. The close intermingling of the fasciculi is such that the interference wherever situated never produces a unocular field defect, except in the earliest stage, nor a pure homonymous hemianopia.

Tract

A lesion here produces homonymous hemianopia which may be congruous or incongruous. Not infrequently the defect is more advanced in one field than in the other, and cases of sector or quadrant defect in one field only due to tract interference have been reported. Tract hemianopia is frequently incomplete and shows variations in intensity in different parts of the defect which often presents sloping margins. The fixation area may or may not be spared. Lesions of the anterior end of the tract may betray their position by affecting the chiasma also, in which case field defects of bitemporal type become added to the homonymous defects already present. It is possible that incongruity may be more pronounced or more frequent in lesions of the anterior part of the tract than in those of the posterior part, but apart from this there is no perimetric sign indicative of the level at which the interference is situated.

Geniculo-Calcarine Path and Visual Cortex

Congruous homonymous hemianopia with sparing of the fixation area is the characteristic field change produced by a lesion beyond the tract. The fixation area is seldom divided. The determination of the site of interference is based on pathological as well as on anatomical grounds since the character of the field changes depends largely on the nature of the lesion. Throughout the path the field changes conform to the anatomical conditions, a superior lesion producing an inferior defect and so on, but the perimetric features do not in themselves indicate the level of the lesion. Here again lesions may affect a considerable length of the path or may be multiple, and in this way difficulty in interpretation may be produced. The subject will be dealt with later in more detail (Chapter XI).

PATHOLOGICAL INTERPRETATION

The pathological interpretation of field defects depends upon the correlation of the visual changes with the nature of the causal morbid process. It is concerned with the onset and course of the changes and with their extent, intensity and uniformity. It is hardly necessary to point out that the pathological interpretation cannot be considered apart from the anatomical since different parts of the nerve path are especially liable to different forms of disease.

In respect of the field changes produced we may regard a lesion in the visual path as consisting of a focus of disease shading off into healthy tissue through a surrounding zone of impaired nutrition due to œdema or congestion caused by the reaction round the focus. In the field these conditions are represented by an area of intense defect surrounded by a zone of less depressed vision. Impaired nutrition leads to weakened resistance so that, if the adverse conditions continue, the focus expands by including some of the surrounding damaged tissue, while previously healthy elements become damaged in turn. If the adverse conditions diminish the contrary process occurs, and only the more severely injured elements remain permanently impaired. (See also Behr 25.)

Some of the characters of defects are more easily explained than others. The extent of a defect corresponds to the amount of the cross-section of the visual path which is damaged, wherever the lesion is situated. On the other hand, although the intensity and uniformity—or absence of uniformity—of defects conform broadly to similar features in the causal lesion, the interpretation of these relationships is not always simple. The severity of the lesion as indicated by functional testing depends on two factors—the number of nerve elements affected, and the degree of damage suffered by each element. It is impossible to differentiate by perimetry between nerve fibres or cells which are divided or killed, and others which are merely temporarily incapable of conducting appreciable impulses. In partial impairment of function colour vision is first diminished and vision for white is demonstrably reduced only when the impairment has reached a certain degree. In the area of a lesion a large percentage of the nerve elements may be slightly affected, the remainder being either healthy or severely damaged. Here the chief element is the preponderance of fibres with impaired conduction as against fibres with normal or no conduction, as might be expected in a lesion which is developing or which is approaching recovery. Vision for white and colour are both reduced but there is an excessive loss of response to colour, that is to say disproportion is present (Roenne 327). This indicates the mobile nature of the lesion and suggests that either further advance or improvement may take place. The greater the degree of disproportion the more rapid the change and vice versa. Disproportion may be present in different degrees at different parts of a defect and the indications it affords are correspondingly applicable. On the other hand there may be in the area of the lesion a small percentage of severely damaged fibres, fairly evenly distributed, all the others being healthy or a few slightly damaged. In this case the main feature is complete loss of conduction in most of the affected fibres, which constitute only a small percentage of the whole, a condition suggesting that all the damage has been done and no more is impending, immediately

at any rate, as might be expected in a stationary lesion. Here proportion between the fields for colour and white is found and the prognosis is little or no further increase but also little or no improvement. The relative numbers and distribution of the healthy, partly damaged and severely damaged elements vary according to circumstances, and the nature of the intensity of the corresponding field defect may justifiably be regarded as always the resultant of the two factors mentioned.

The relation between the uniformity of the defect and that of the lesion is complicated by the varying value of the field between centre and periphery, and by the question whether functional impairment is as easily produced in one set of fibres as in another by the same degree of damage. Reference has already been made to the possibility that some cases of zonular ring scotoma, for example, may be due to the pronounced superiority of visual acuity at the fovea over that of its immediate neighbourhood. The point is of relatively slight and largely theoretical importance, and only arises in relation to parts of a defect at different distances from the fixation point. Although more accurate knowledge would help to reduce our margin of error in making inferences, there seems no reason to fear that this will be excessive if we assume that in any field defect different degrees of visual loss represent areas of damage in the visual path correspondingly differing in severity.

Field changes which suddenly appear or alter indicate correspondingly active lesions. When the defects are found to change rapidly in position, shape, extent or intensity, it may be inferred that the lesion in the nerve path is of acute type, inflammation or rapidly growing tumour, for example, while comparatively stationary fields indicate more chronic lesions.

The visual neurones are extremely sensitive to impaired or vitiated nutrition. This may result from restricted blood supply or congested venous circulation produced in various ways by such influences as mechanical pressure, disease, injury or cicatrization affecting the nerve path or the adjacent tissues, or by the presence of toxins of exo- or endogenic origin.* Moreover, since the sense of sight is easily tested, slight variations in function can be more readily discovered than in the case of other nerve mechanisms. For this reason the visual nerve path is perhaps in some ways specially suitable for the clinical study of impaired nerve function (Uthoff 418).

As regards the photochemical apparatus, alterations in nutrition are brought about in several ways. Inflammatory foci in the choroid or defects in the choroidal or retinal circulation may lead to oedema, or to cicatricial or degenerative changes in the retinal outer layers. The rods and cones may become loosened from the pigment epithelium as in the initial stage of detachment from whatever cause. A general lowering of nutrition, or vitamin deficiency, as in war hemeralopia or xerosis conjunctivæ with hemeralopia, can apparently affect the retinal outer layers, though here the difficult region of what is called functional disorder is approached. To what extent toxins act directly upon the rods and cones is unknown; the night-blindness of jaundice is probably due rather to the absence of fat-soluble vitamin than to the presence of toxic substances.

* *Vide* the well-known physiological experiments in which conduction is blocked by exposure of a section of a nerve to alcohol vapour, CO₂, oxygen deprivation, etc.

There is evidence also that damage or functional impairment may result from an over-exercise of the normal metabolic processes such as is induced by excessive light. Direct damage may be inflicted by tumour growth, but the visual results of this injury on the outer layers have not been studied. Lastly, primary degeneration may occur, the choroidal circulation being apparently intact.

The clinical manifestation of these conditions is depression of the field, exaggerated in dim light, and relative yellow-blue blindness, probably due to an insufficient or sluggish provision of visual purple together with some impairment of the cones. Ring scotoma of zonular type is a common feature. Blue appears "pale" or greenish or green, and some observers have found the perception of yellow to be diminished. If the conditions of the test are made suitable, impairment of vision for red may also be elicited. If the lesion progresses and the damage becomes more severe, the impairment for red becomes more prominent, so that the distinctive yellow-blue blindness is submerged in the intensity of the defect. It is, therefore, characteristic of early or partial impairment and not of severe damage or complete destruction.

In many affections of the outer layers the power of recovery is extremely high in relation to the loss of function at the stage of greatest intensity provided that the initial severity of the condition has not been too great or its duration too prolonged. Though highly specialised the cells are very resistant to injury.

As far as concerns the special features of the field changes produced by outer layer impairment, perimetry alone can do little to distinguish the various causes, and the interpretation of defects is mainly directed towards the anatomical aspect, that is, to the determination of the site of the interference.

In the case of the conduction apparatus, which is directly nourished by blood-vessels, impairment of nutrition may occur in the form of restricted arterial supply or obstructed venous return. Both factors may act together. Pressure, inflammation, vascular disease and injury and their results, both direct and indirect, are common causes. To toxic substances whether of exo- or endogenic origin the retinal ganglion cells and their fibres as far as the chiasma seem specially susceptible. The first result of impaired nutrition is diminished conducting power which leads to a general lowering of perception affecting both white and colour. In its early stages this defective perception is most easily demonstrated by testing with colour such as red or green. It must be remembered, however, that this is not because the first result of impairment of conduction is reduced ability to recognise red, but because the test with red is a convenient clinical method which elicits the alteration in function more easily than a test with blue or white. The defect for red is merely a part of the general impairment of perception and can also be elicited for white and blue if sufficient trouble be taken. Nor has defective perception of red and green any special significance as regards the cause of the conduction impairment, whether by toxins or otherwise. Hæmorrhage or œdema between the nerve fibres does not influence conduction *per se*, hence the good vision present in papillœdema. When the œdema begins to be replaced by cicatricial tissue secondary atrophy and visual impairment appear. The defective area corresponds in degree and extent to the intensity and distribution of the interference. The nerve fibres possess a high power of

resistance, and, provided that the adverse conditions have not been sufficiently severe or prolonged, restitution of function may ultimately ensue after a considerable period of complete abeyance.

Should the injurious influence act with sufficient intensity or for a sufficient time, death and destruction of the axones occur, rendering recovery impossible. The visual defect is now permanent and is not always associated with a proportionate alteration in the colour of the optic disc, so that the amount of atrophy present can only be correctly estimated by perimetry.

Field changes due to conduction impairment may therefore be regarded as all fundamentally of the same nature and as the expression of deficient or vitiated nutrition somewhere in the visual path.

Apart from these general considerations, the pathological significance of the different forms of field defect is in many ways restricted. It is not possible in the present state of our knowledge to identify special forms of defect with individual diseases to more than a limited extent. A certain relationship of defects with morbid processes can nevertheless be traced. Disease spreading inwards from the pial sheath along its trabeculae gives rise to peripheral depression of the field in tabes, as a result of plerocephalic* oedema of the nerve (choked disc) and in certain forms of retrobulbar neuritis. The field changes thus produced present differences as well as similarities. Homonymous hemianopia may be due to hæmorrhage, thrombosis or tumour, and here also differences in such characters as onset, intensity, and course are of value in diagnosis. Neither the common centro-cæcal scotoma nor the arcuate bundle defect originating in the nerve corresponds to a special site or a special disease, but the circumstances under which they occur suggest that the former may be primarily due to toxic and the latter to vascular causes arising from pressure or inflammation. At the same time the field changes of tobacco amblyopia and glaucoma are almost pathognomonic, and it will be found that, when all the characters of field defects are collectively as well as individually examined, similar varieties due to different causes often present differences of various kinds which may help to establish their identity, or at least to suggest some form of classification on a pathological basis.

Affections of the conduction apparatus may be arranged in four main groups:—

I. Toxic and Inflammatory Conditions

These affect the subgeniculate, mainly the subchiasmal, portion of the visual path and include retrobulbar neuritis and the toxic amblyopias.

The field defects vary but are predominantly central, the onset is usually rapid and the tendency to recovery is almost always a prominent feature. In certain types variation and fluctuation of the field changes occur. The causation of the field defects is complex and their characters are probably related to the minute anatomy of the terminal nutrient vessels and depend largely on specific toxophile tendencies of the nerve elements, and on vascular congestion and obstruction due to inflammation. Probably all these

* See footnote, p. 143.

causes act together in varying proportions in different cases. It is noteworthy that nerve fibre bundle defects are not found in impairment of vision due to exogenic poisons such as tobacco, quinine or wood spirit.

II. Pressure

Any part of the path may be affected, the optic disc (glaucoma) and the chiasma (tumour) being common sites of incidence. The defects involve both centre and periphery of the field, usually come on gradually and are progressive leading to blindness if relief is not obtained. Variation in the pressure is accompanied by changes in the field defects and removal of the pressure is followed by rapid recovery unless it has lasted too long. The effects are produced by venous congestion and arterial ischaemia, rather than by direct compression of the nerve fibres. Plaque formation similar to that which occurs in retrobulbar neuritis has been found in cases of pressure on the optic nerve.

In all cases of slowly advancing field defect care should be taken to distinguish the pressure type from the inflammatory type. A careful study of the history of onset is frequently very helpful.

III. Vascular Disease

Hæmorrhage, thrombosis and angiospasm occur in the geniculo-calcarine path and in the retina and anterior end of the nerve; less frequently in the posterior part of the nerve, the chiasma and tract. Embolism has been reported in the central retinal artery and probably occurs elsewhere. The onset of the field defect is usually sudden and the degree of recovery usually slight except in angiospasm, in which it may be complete on one or more occasions until an attack occurs which causes permanent damage. Hæmorrhage near, but not into the pathway, may produce a pressure defect terminating in recovery by absorption. The shape and position of the defects depend on anatomical factors at the site of interference, the onset, intensity and course on the nature of the disease.

IV. Traumatism

Fracture of the base of the skull and injuries of the occipital region are the most common causes of traumatic lesion of the visual path. A certain amount of recovery takes place corresponding to the areas in which the damage is not irreparable, as in the case of œdema surrounding a cortical wound or temporary congestion or hæmorrhage in the optic foramen. A permanent defect remains corresponding to the nerve elements whose nutrition is damaged or cut off by the rupture of vessels or by their subsequent obstruction through cicatricial processes. The defects produced are determined by the anatomical conditions at the point where the injury takes effect. As is to be expected, central defects of this kind are often associated with peripheral defects when the lesion is sub-chiasmal, but may be isolated when the lesion is cortical as here the central elements may be affected alone.

In almost all forms of interference with the visual path, the tendency to recovery when the cause is removed or ceases to act, which has already been referred to, is a

prominent and important feature. It is pronounced in acute inflammatory cases, in which complete blindness may eventually be replaced by practically normal vision, and is well marked in pressure cases in which vision is sometimes restored in an area which has been blind for months. In certain cases this tendency is less in evidence, either owing to the severity of the initial lesion, or to the development of secondary changes leading to loss of nutrition. As a rule the longer the functional loss persists the less is the chance of ultimate restitution—in other words, the prospect of recovery can be estimated better by the duration than by the severity of the visual defect. The tendency to recovery is apparently independent of the causal factor ; it is present, though not always in the same degree, in toxic, inflammatory and traumatic conditions (unless the initial lesion has been too severe, *i.e.*, destructive), and indicates that while the function of the nerve elements is easily impaired their vitality is relatively high.

It will be noted that the various parts of the visual path are associated with different morbid processes in a fairly well-defined manner. The optic nerve, which alone possesses a definite fibrous trabecular framework, has almost a monopoly of toxic and inflammatory conditions which become much less common at a higher level. Syphilis and multiple sclerosis, however, attack the chiasma and lower part of the tracts occasionally. In civil life traumatism most frequently affects the nerve, rarely the chiasma. Pressure and vascular disease are relatively uncommon in the nerve.

At the chiasma pressure interference from tumour, usually hypophysial, is by far the most common lesion, syphilis and multiple sclerosis are much less frequent, and other conditions rare. The tract is less often affected than the chiasma ; the causes of interference are similar, but pressure from outside, *e.g.*, from temporal lobe tumours, becomes relatively more frequent.

In the geniculo-calcarine pathway the nature of the morbid conditions again changes. Vascular lesions of various kinds are far more frequent than any other. Tumour pressure is by no means uncommon, but in relative frequency comes far behind vascular disease. Toxic and inflammatory lesions, apart from meningitis and abscess, are exceptional. In the occipital region traumatism appears again, but in civil life only very occasionally.

Functional Field Changes

Another group of changes results from functional or psychical causes. Varying or uniform depression with spiral or tubular fields, incompatible with organic lesions and with other features in the clinical picture, are characteristic. Such defects are sometimes superimposed on others of organic origin. These field changes will be discussed in Chapter XII.

Lastly, the possibility of a certain degree of normal variation, perhaps more pronounced in pathological cases, must be borne in mind. Slight alterations may be found when a case is being periodically examined from time to time and should be interpreted with caution. Practice increases the field and a slight enlargement in the case of a patient, who has been examined several times and is becoming a good perimetric subject, does not necessarily mean an improvement in the morbid condition present. On the

other hand, if the patient feels out of sorts or is disinclined for the test, defects may appear exaggerated although the disease is no worse.

We see, then, that the interpretation of field changes presupposes accurate observation, and is based on the correlation of all the characters of the defects with anatomical, physiological, pathological and psychical factors.

Summary of the Characters of Defects and their Relation to Interpretation

Anatomical Interpretation	1. Position.	Indicates the position of the lesion in the cross section of the visual pathway.
	2. Shape.	Indicates the shape of the cross section of the affected area in the pathway modified by the mode of distribution of the fibres.
	3. Size.	Indicates the extent of the lesion in relation to the cross section of the pathway.
Pathological Interpretation	4. Intensity.	Indicates the degree of damage done to the nerve elements.
	5. Uniformity.	Indicates the relative amount of damage in different parts of the area of the lesion.
	6. Margins.	Indicates the sharp or graded nature of the edge of the lesion.
	7. Quality.	Indicates outer layer interference in retina.
	8. Behaviour.	Indicates the onset and course of the lesion and thus points towards its nature.

In connection with diagnosis and prognosis and in estimating the results of treatment an opinion should be based on the study of *all* the characters. It should always be remembered that it is the type of field change, not the size of a field defect, which is of consequence.

CHAPTER VII

THE CHOROID AND RETINA

THE previous chapters provide us with some equipment for a more detailed consideration of the chief conditions in which perimetric examination is useful. It will be convenient to begin with the retina and to discuss seriatim the field changes associated with lesions of the different parts of the visual path up to the occipital cortex.

For the most part choroidal or retinal lesions are easily visible ophthalmoscopically, and the study of the field defects, though interesting and often valuable, is not always indispensable as regards diagnosis and treatment. The visible changes may, however, be disproportionate in one direction or another to the degree of visual impairment, or the ophthalmoscopic examination may, for various reasons, give doubtful or inconclusive results or may require confirmation. In such cases perimetry is often very helpful.

Retinal affections may be divided into those in which the outer and those in which the inner layers are chiefly concerned. On the other hand, the lesion may not be confined to either layer, and some conditions are more suitably dealt with elsewhere. The toxic amblyopias have, therefore, been placed with affections of the optic nerve, and glaucoma is referred to separately.

I. CONDITIONS MAINLY AFFECTING THE OUTER LAYERS OF THE RETINA

Choroidal lesions involve the retina secondarily, and especially the outer layers. The field changes produced present the features of damage to the photo-chemical apparatus, relative yellow-blue blindness, as long as the function of the outer layer is only impaired. With more severe damage the special characters of the defect become lost in its greater intensity and the perception of both red and white test-objects is destroyed or reduced to such an extent that a comparison test between blue and red gives uncertain results. As patients often apply for advice only after the condition is well advanced, the special quality of the defect is not always found in clinical practice. Permanence of a certain degree of yellow-blue amblyopia has been reported and presumably indicates that the lesion has remained partial.

Disseminated Choroiditis

The changes are easily seen with the ophthalmoscope and perimetry is not often undertaken.

In a recent case some general depression of the field is usually present, especially if the changes are widespread. Patchy scotomata, corresponding approximately in extent to the visible lesions, are found, which may show, in the early stage, the characteristic relative yellow-blue blindness in greater or less degree, according to the intensity of the defect.

When associated œdema of the nerve-head is present enlargement of the blind spot, sometimes very pronounced, may be found. The correspondence of the field defects

to the visible lesions shows that the nerve fibre layer remains functionally active, without touching on the question as to whether or to what extent it is involved in the inflammatory process. Nerve fibre bundle defects are not common in ordinary disseminated choroiditis, though, of course, they may occur if the lesion spreads through the retina. They will be referred to later.

It is often difficult to correlate the degree of visual loss with the ophthalmoscopic appearances, which may suggest much better or much worse functional efficiency than is actually present. The presence of gross field defects with severe visual loss indicates, in the earlier stages at any rate, functional interference rather than serious organic damage, and, therefore, does not necessarily point to a bad prognosis. A high degree of recovery is common and often surprising in relation to the state of the vision when at its worst. Persistence of the defects is of worse prognostic significance, and it would appear to be the duration rather than the severity of the visual changes which should be regarded as a reliable guide.

In the mature and permanent stage, when the condition has become a disseminated choroidal atrophy, the stage most frequently seen in practice, the defects are more definite and more intense. Relative yellow-blue blindness is no longer found, and the nerve fibre layer still escapes. Here also, good vision by Snellen's types may be associated with gross visible changes, central as well as peripheral, in the fundus. In such cases the scotoma is not infrequently ring-like in character, or vision may be retained in a small area at, or close to, the fixation point, illustrating the very local nature of the process and the high resisting power of the foveal cells.

Ring scotoma is a characteristic feature of disease of the outer retinal layers and is prominent in syphilitic choroido-retinitis and in retinitis pigmentosa. In the former condition Ole Bull found complete or partial, sometimes irregular, wing-like or crescentic, arcuate or ring scotomata, which he regarded as practically pathognomonic of syphilis. A certain number of these are probably nerve fibre bundle defects due to damage to the inner retinal layers from the associated disease of branches of the central retinal artery. In recent cases they tend to be variable and disappear on recovery. Baas pointed out that in syphilitic central choroido-retinitis, the scotoma, though very minute, may be of the ring variety.

In visual impairment, due to macular disease, whether primarily choroidal or retinal in origin, ophthalmoscopic changes are practically always seen, and perimetry is not often employed. The retinal lesions are, however, not always easily visible even with the most careful examination, and in such cases, especially when both eyes are affected, either simultaneously or in more or less rapid sequence, other conditions may be simulated and the diagnosis may be, for a time at least, extremely difficult. The difficulty is increased when the field defects suggest homonymous or bitemporal hemianopic or quadrant-shaped scotomata. This may be the case without the presence of visible corresponding retinal lesions, for it is by no means always possible to correlate the field defects with the visible changes. The latter may be less discernible in the eye in which the field defects are more pronounced and vice versa. Even when minute retinal lesions are detected the extent of the field defects often indicates a much larger area of

functionally altered retina than that in which changes can be seen by the ophthalmoscope. Metamorphopsia may not develop until later. Ring scotoma may be present in the initial stages.

There may be sufficient swelling of the optic disc to suggest retrobulbar neuritis or even increased intracranial pressure. Disease of various kinds affecting the nerves, chiasma, or tracts (Leber's disease, disseminated sclerosis, tumour) may be suggested.

In other cases the scotoma may be very small, less than 2° in diameter, and of very low intensity.

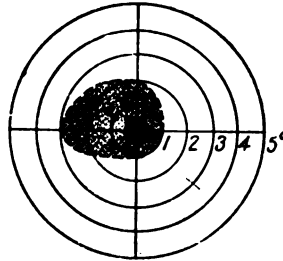


FIG. 34.—CENTRAL CHOROIDO-RETINITIS.

Scotoma 1° in diameter for $20/20$ white, and 4° for $20/20$ red. Vision $\frac{6}{36}$ plus. (P. 1912.)

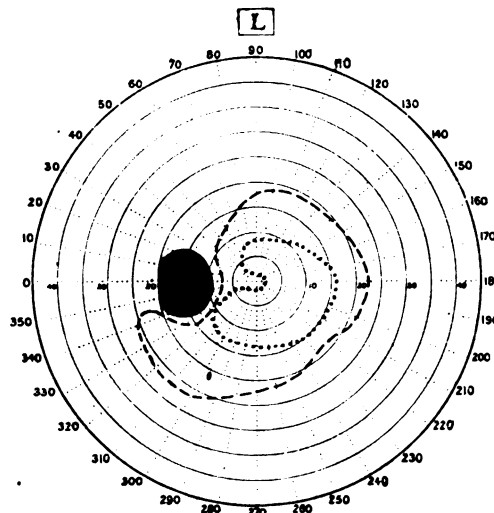


FIG. 35.—CENTRAL CHOROIDITIS WITH GROSS SWELLING OF THE OPTIC PAPILLA, SIMULATING UNILATERAL PAPILLOEDEMA
Objects: Blind spot $20/20$. Broken line $20/20$. Dotted line $20/20$ red and blue. Vision $\frac{6}{36}$. (B. 1925.)

The screen test at not less than 2 metres distance affords the best means by which such defects can be adequately examined. They differ from defects due to nerve lesions in being more vague and indefinite in character, and when hemianopic in type the vertical meridian is not strictly followed. Relative yellow-blue blindness may be elicited. The somewhat indeterminate character of these changes stimulates the observer to a specially careful scrutiny of the retina which usually enables the diagnosis to be established.

Choroidal Changes in Myopia

Much has been written in regard to the field of vision in myopic eyes by Weiss and other observers. Peripheral contraction has been noted, but in itself has little special

significance or value as a clinical sign. Multiple concentric ring scotomata have been described and may be regarded as of functional origin. More important are the alterations in the central field which occur as paracentral or central defects of various kinds associated with visible choroidal changes. Different forms of juxtacæcal scotoma may be present, including arcuate and annular defects. Most, if not all, of these appear to be of zonular type due to the distribution of the choroidal degeneration, and conduction interference with groups of nerve fibres does not appear to occur. Typical nerve fibre bundle defects with nasal step have not been found in uncomplicated choroidal atrophy in myopia.

As a rule the surgeon obtains all the information he requires by the ophthalmoscope, but in certain cases this examination may advantageously be supplemented by perimetry, especially when the question of the onset and progress of central degeneration arises. According to Cantonnet functional alteration precedes the visible changes in the area between the disc and the macula. When choroidal degeneration is seen commencing at the outer side of the disc examination with the screen will show whether the associated defect corresponds to or is in excess of the visible changes. In the latter case, especially if the edge of the scotoma towards the fixation area is sloping or indefinite, a progressive choroidal degeneration is indicated, whereas, if the defect corresponds fairly closely to the area of visible change and presents a sharp margin towards the centre of the field, the process is relatively stationary.

Visible changes may also be present in or near the macular area, although central acuity by Snellen's test is still good, and in other cases in which the fundus is not clearly seen it may be desirable to ascertain the state of the macular region. In such cases the screen examination with small white or coloured tests is helpful. In commencing choroidal atrophy the test-object is seen to "go in and out" and although foveal vision begins to decline, it is often not at the fixation point that the defect first becomes manifest. Thus the atrophy may gradually advance without disturbing the patient until the fovea becomes involved, when the loss of central acuity immediately causes anxiety. In some of these cases the examination of the central field assists the surgeon in regard to prognosis and guides him in advising the patient.

Traumatism

Although in traumatic rupture of the choroid a crescentic scotoma corresponding in shape and position to the lesion might be expected, I have hitherto failed to demonstrate a defect of this kind, nor is there any evidence of damage to the nerve fibres at the site of the rupture. A central scotoma of retinal outer layer type is present, due to the associated macular disturbance.

The condition known as *commotio retinae*, in which the macular area presents a more or less œdematous appearance ophthalmoscopically, usually affords an excellent demonstration of the relative central scotoma for blue with well-preserved perception of red. There may be also a scotoma for white, sometimes very small, according to the severity of the macular injury.

Where the qualitative colour scotoma alone is present the prognosis is good as this

defect disappears in a few days ; the scotoma for white indicates the probability of permanent impairment of vision.

Detachment of the Choroid

Two varieties of this condition are recognised, the post-operative and the spontaneous. In post-operative cases perimetry is not usually required for diagnosis or prognosis nor are the circumstances usually suitable for this form of examination. Although the retina retains its relationship to the choroid, functional impairment of the affected area is severe. Vision over the detached area is much more depressed than over an area of shallow detachment of the retina, in which the retina is separated from the choroid, and it is more depressed than over a flat solid tumour in which case the retina is in contact with the choroid. The explanation may possibly be that the great elevation of the choroid interferes with its vascular supply. However this may be, vision returns in post-operative choroidal detachment when the choroid resumes its position. Little is known of the details of the state of the field. Fuchs found that the field was frequently contracted for ordinary perimeter tests, though large objects could be seen as far as the normal boundary. In one case he noted that the contraction for blue was extreme in comparison to that for white. In several cases examined by the author the visual defect was much more intense, so that differential tests could not be carried out. The prognosis is good and the question of differential diagnosis is unlikely to arise.

In the spontaneous form there may be true detachment of the retina in addition at one or more points, and the diagnosis may lie between simple retinal detachment, choroidal detachment and choroidal tumour. In some recorded cases retinal function has been found to be retained longer than in retinal detachment, and in one reported by Simon the diagnosis was assisted by perimetric examination, which showed that colour vision and adaptation were preserved except over the parts where the retina was separated also. The spontaneous form is uncommon and the prognosis unfavourable. Vision does not always return, should the choroid resume its position, probably owing to the presence of disease.

Retinitis Pigmentosa

The field changes are characterised by the presence of a ring scotoma, peripheral depression, slow progress and the relative preservation of central vision. In addition, night blindness is present in greater or less degree, and the perimetric defects are exaggerated in dim light.

A systematic detailed analysis by the quantitative method does not appear to have been carried out as yet. Bjerrum, however, found that the field periphery, when apparently normal for large objects, showed considerable restriction with a small test, and Köllner elicited perception in the periphery by strong stimulation when the defect was apparently absolute when tested by the customary methods. The essential field change is therefore a gradual depression, commencing first in the inner part of the peripheral zone, then involving the periphery, and lastly, as a rule, the centre. Various forms of field change have been described, but it is preferable to regard the different types of defect as stages in a progressive process.

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In the early stages widespread irregular and uneven depression of the field appears except in the centre. Especially in the inner part of the peripheral zone patchy indefinite scotomata are found, difficult to map out exactly. Central vision may be $\frac{6}{6}$ with a field for $\frac{1}{2000}$ or $\frac{2}{2000}$ of only a few degrees in diameter. This small central field persists while the changes develop in the more peripheral parts. The patchy defects coalesce to form a ring scotoma which is usually well developed by the time that the patient seeks advice.

This scotoma lies in the outer part of the central area and the inner part of the peripheral zone, usually between the 10 and 40° circles. Its width varies approximately from 30 to 60°, and, at first, the intensity may be slight and the ring incomplete. The intensity varies so that in some parts the depression may be slight and in others severe or absolute, and on this account a complete ring may appear partial unless the examina-

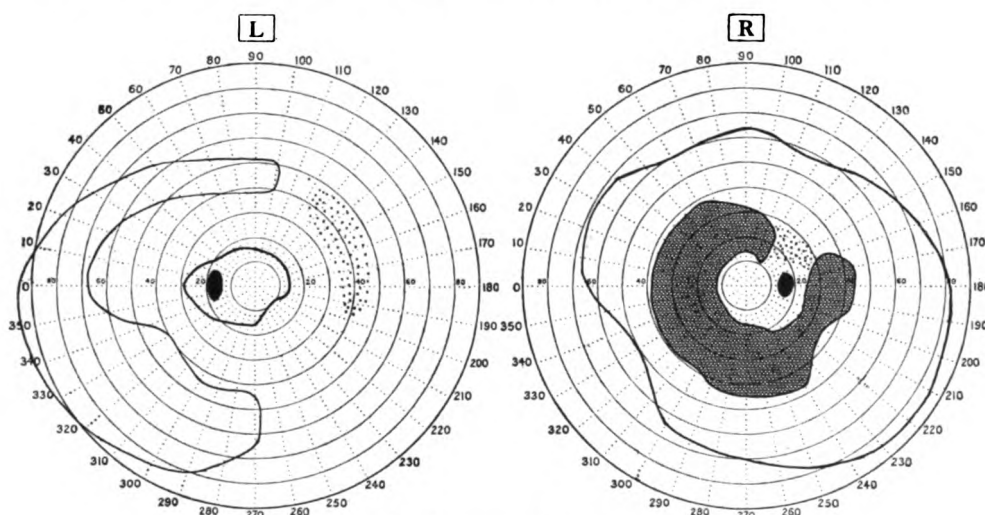


FIG. 36.—RETINITIS PIGMENTOSA.

Loss of nasal field in left eye ; same developing in right. Ring scotoma. Object $33v$ dimly seen in dotted areas. $V = \frac{6}{36}$ R. and L. (C. 1919.)

tion is thorough. The margins are rather steep centralwards, but more sloping peripherally. Associated with the ring scotoma is a certain degree of peripheral depression. As the disease progresses the scotoma becomes denser and wider, more by peripheral than by central extension, and at the same time the peripheral depression becomes deeper, so that finally the scotoma breaks through to the periphery, usually towards the nasal or upper side. The peripheral field now begins to disappear at the same part, so that in the later stages only a sickle-shaped temporal or lower temporal patch remains, requiring a strong stimulus for its demonstration. Large and bright test-objects, such as a sheet of white paper, should be used in testing the peripheral retina before concluding that it is absolutely functionless. The central field is usually round, but may be a somewhat horizontal oval, extending farther temporally than nasally. At first it extends some 30° to 10° all round the fixation area, but, as time goes on, slowly becomes more and more restricted and very steep-edged. It retains its acuity in a relatively high degree to a late stage, even when extremely contracted, and nearly always survives the

peripheral remnant. The steep edges of the small central field are largely responsible for the loss of orientation which is so troublesome to patients with retinitis pigmentosa.

Colour vision is lost in proportion to vision for white in accordance with the very slowly progressive nature of the lesion. A special yellow-blue amblyopia has been noted, but is not a commonly observed feature, probably on account of the stage at which the cases usually come under observation.

The defects progress slowly and gradually and incline strongly towards symmetry in the two fields, although they may be more advanced on one side than on the other. Exaggeration in dim light, the so-called *torpor retinæ*, is a prominent feature throughout.

Variations from the typical characters have been described, but are uncommon. The ring scotoma may increase chiefly by centripetal extension leaving a relatively extensive peripheral seeing zone, and sometimes the central field may be lost before the outer part has completely failed. Central scotoma is also reported, but is rare.

The field changes correspond approximately to the pigmented area of the retina, the chief difference being apparently in the direction of excess of field defect over visible pigment.

In early cases, in which little or no pigment can be discovered (retinitis pigmentosa *sine pigmento*), the ring scotoma is present, and its character assists the differentiation from other varieties of night-blindness without retinal changes such as the congenital hereditary non-progressive form, in which the fields are contracted especially in feeble light, and from functional cases.

An ophthalmoscopic picture resembling that of retinitis pigmentosa may be present in cases of retinal pigmentary degeneration secondary to chronic choroidal disease, but numerous scotomata, varying in shape and size, are distributed throughout the field seldom forming an actual ring scotoma, and never as the first sign, though they may be more numerous in the intermediate zone. These scotomata appear to remain stationary for a long time, and the peripheral contraction is slight (Hepburn, 172). Hepburn regarded the perimetric findings as useful in differentiating between these two types in cases in which the diagnosis on other grounds is doubtful.

Prognosis is guided by the fact that the central field usually outlives the peripheral. As long as there is some vision in the periphery, complete central loss is in most cases still some distance ahead.

Beyond providing additional evidence that in the first instance it is the retinal outer layers which suffer, perimetric studies have done little to throw light upon the pathology of the disease. The nature of the field changes and their onset and course are definitely against the view that the disease is based upon an equatorially situated failure in the choroidal circulation.

Other Varieties of Ring Scotoma due to Impairment of the Outer Layers

Ring scotoma may be due to bright light or to malnutrition. These cases form a somewhat indefinite group in which the distinction between actual injury at the one extreme and a psychical manifestation at the other is often obscure.

Examples have been reported following exposure to lightning flash (Hancock),

and to furnace light (Claiborne). Diffuse bright light was apparently the cause in the cases of fliers and anti-aircraft artillerists observed by Zade, who found narrow, relative, annular or sickle-shaped defects peripherally situated usually about 50° , rarely less than 35° , from the fixation point. Such scotomata were temporary, but sometimes remained for several months and were avoidable if dark glasses were used. Somewhat similar scotomata were found by Speleers and Jess in cases of visual damage by sunlight.

Ring scotomata of this kind are, with possibly a few exceptions, functional in origin and are distinguished by their very narrow width, peripheral position and progress towards recovery when the cause is removed.

In night blindness due to malnutrition or war conditions ring scotoma has been found. Perimetry has proved of service in separating those cases due to definite choroido-retinal disease from the—under campaign conditions—commoner form due to functional impairment, an important matter in connection with treatment and the allocation of duty.

A low degree of night blindness is often present in myopia, and ring scotomata have been found (Usher), but their causation is not thoroughly established. Apart from choroidal disease they are probably psychical manifestations.

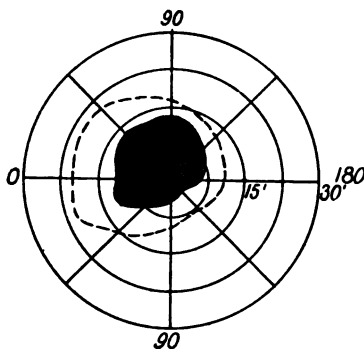


FIG. 37.—ECLIPSE SCOTOMA.

Each circle represents one-eighth of a degree. Scotoma for $\frac{3}{4000}$ red (broken line), with centre for $\frac{1}{4000}$ white. Maximum diameter just over 0.5° . Vision $\frac{5}{6}$ plus. Slight macropsia present. (W. M. 1912.)

Eclipse Scotoma

Retinal lesions following exposure to bright light such as the sun, electric welding, or electric flash are probably due to the effects of heat. A small central scotoma is the usual result though ring scotoma, as already mentioned, has been recorded.

The most common of these conditions, and the one most studied, is the eclipse scotoma which follows unprotected observation of an eclipse of the sun. Vision by Snellen's types is often good, though symptomatic of central scotoma, and ophthalmoscopic examination may be negative. The scotoma is usually very small, rarely over a degree and often less than half a degree in diameter, and may be exactly central or, more commonly, may extend rather more to one side of the fixation point than to the other. It is relative or relative with an apparently absolute centre, the loss for colour being more extensive than for white. A permanent impairment of vision for yellow has been recorded (Mackay). While the diagnosis is usually easy the examination of the scotoma provides additional evidence, which is always helpful, and sometimes essential.

A large and dense scotoma indicates a relatively bad prognosis, although a high degree of recovery, except in the worst cases, is the rule. When recovery appears to be complete, the actual degree of restitution should be investigated by the screen, for vision may reach $\frac{6}{6}$ with ability to read print with fair fluency, while a small scotoma remains.

Detachment of the Retina

The perimetric evidence of detachment of the retina consists of visual loss varying in intensity and corresponding in extent and position to the projection of the separated area.

The defect is usually relative at first, and after some time becomes absolute. It merges into the seeing field by a sloping marginal zone of varying width and gradient according to the nature of the case. A sharp edge may be mechanically produced by an overhanging fold of ballooned retina, and the transition is usually less abrupt in recent than in old cases.

An interesting feature is the relationship of the field for blue to that for red. When the retina is elevated by serous fluid the resulting defect for blue is relatively more intense than that for red, so that the red field overlaps the blue, producing a true interlacing of the colour fields. Blue appears green or greenish, whereas red is correctly seen, presumably on account of the impaired nutrition of the outer layers, and perhaps also because of the separation of the cones from the pigment cells by a layer of yellowish fluid. This overlapping is often best elicited in the central field where colour perception is normally most acute, and is more easily found in recent cases in which the retina is still functioning fairly well. It was regarded by Treitel as pathognomonic of detachment, especially if present in a circumscribed area. According to Köllner, relative blue blindness (acquired tritanopia) occurs in other conditions in the central area of the field, and is rarely an *early* symptom of detachment. I have observed it at a very early stage.

The examination of the field defects in detachment is of value : (1) In assisting the diagnosis where ophthalmoscopy is difficult or inconclusive ; (2) in estimating the extent of the separation ; (3) in estimating the degree and the uniformity of the functional loss of the separated retina ; (4) in discovering the possible presence of a second lesion ; and (5) in determining complete healing with functional restoration.

In the great majority of cases the diagnosis can be established by ophthalmoscopic examination alone but, while this method can usually easily detect the area of visible or gross separation, it may leave the observer in some doubt if the detachment is very shallow or the media obscure. As a preliminary test, the confrontation method, carried out first in daylight and then in reduced illumination, is very useful, and the elicitation of a defect in the latter case, doubtful or absent in the former, is most suggestive. The perimeter and screen help to establish the diagnosis and show more truly the exact extent of the lesion, especially if the examination is conducted with small white test-objects and with colours. The defective area found by small white and coloured tests is always larger than that found by an object of moderate size, and often more extensive than the ophthalmoscopic appearances would suggest.

By using a series of test-objects, the degree and uniformity of the functional impairment may be estimated, features which may be significant in certain cases.

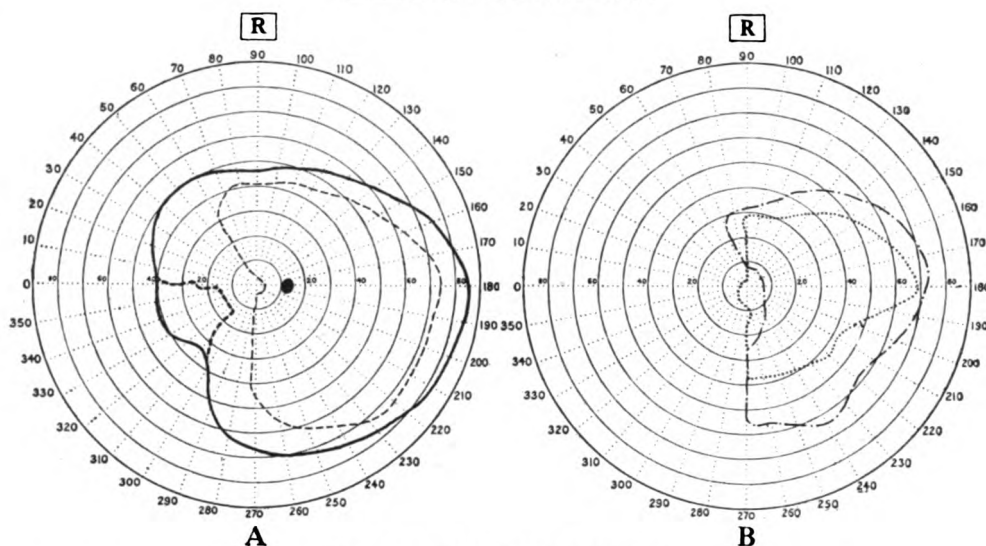


FIG. 38.—DETACHMENT OF RETINA.

- A. Isopters for 7.5 , 5 , and 1 . Field for 7.5 coincides with that for 5 , except on nasal side. $V = \frac{6}{38}$.
 B. Isopters for red and blue $\frac{1}{30}$ from same case, showing relative central defect for blue. True colour interlacing. Red . . . Blue - - - - - (A. D. 1912.)

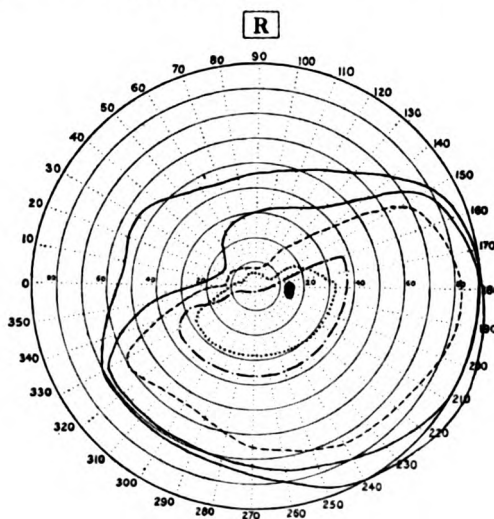


FIG. 39.—DETACHMENT OF RETINA.

- Isopters for 3.0 , 3.5 , and 1.0 white and $\frac{2.0}{30}$ red and blue. $V = \frac{6}{12}$.
 Red . . . Blue - - - - -. Relative central defect for blue. True colour interlacing. (A. C. 1923.)

While we may hope for the development of some method by which the earliest signs of the onset of detachment, at least in those cases in which the onset is gradual, may be demonstrated, it is doubtful, having regard to the pathology of detachment, whether the detection of an area of impaired vision or the failure to detect any such area in a case in which the possibility of future detachment was suspected, would be of definite prognostic significance. Nevertheless, in such eyes, the field should not be neglected, as the perimetric findings form a valuable part of the clinical picture.

After treatment the examination of the field by the confrontation method is of

value in estimating the success of operation and later, when reattachment has apparently occurred, the field should be thoroughly tested by serial objects, colours, or in reduced light, before forming an opinion as to the degree of functional recovery.

Retinal Detachment secondary to Choroidal Tumour

Although in most cases of detachment associated with choroidal neoplasm the

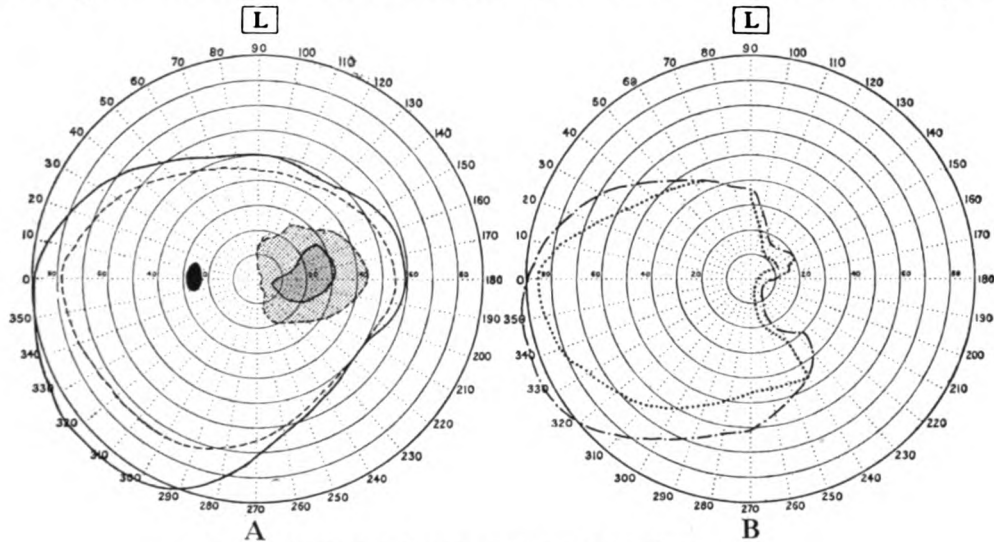


FIG. 40.—CHOROIDAL TUMOUR WITHOUT FLUID DETACHMENT.

A. Scotoma corresponding to area of tumour only. Objects $3\frac{5}{30}$, $3\frac{3}{30}$. V.c. + 4.5 D.S. = $1\frac{6}{8}$.
 B. Isopters for $3\frac{2}{30}$ blue and red in same case. Complete nasal defect; no interlacing. (G. S. 1912.)

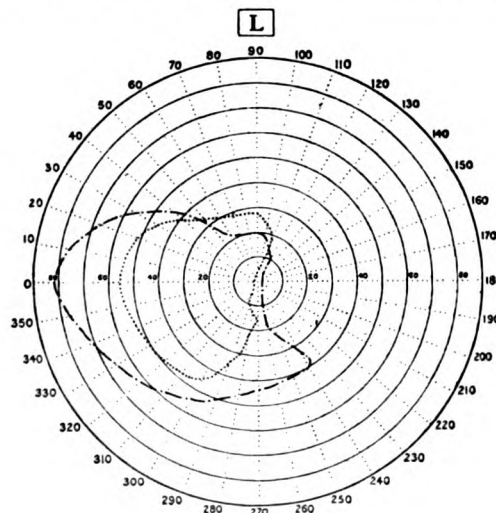


FIG. 41.

Same five days later, $3\frac{5}{30}$. Interlacing above corresponding to appearance of fluid detachment below tumour.
 Red . . . Blue - - - - - (G. S. 1912.)

growth is visible and the diagnosis is comparatively easy, the study of the field changes is not without interest, and a perimetric distinction between the so-called idiopathic

detachment and that due to tumour would be of value to the clinician. In the former the retinal separation is an end result in a diseased eye, whereas, in the latter, the choroid and retina are healthy apart from the local effects of the tumour. In tumour cases the retina is first of all elevated by the neoplasm, but not detached, and, to begin with, no recognisable functional disturbance is produced. As growth progresses visual loss appears owing to interference with the retinal outer layers, though little if at all with the nerve fibre layer, and finally the tumour emerges through the thinned and stretched retina. At one stage or another serum is exuded from the choroid, and, in

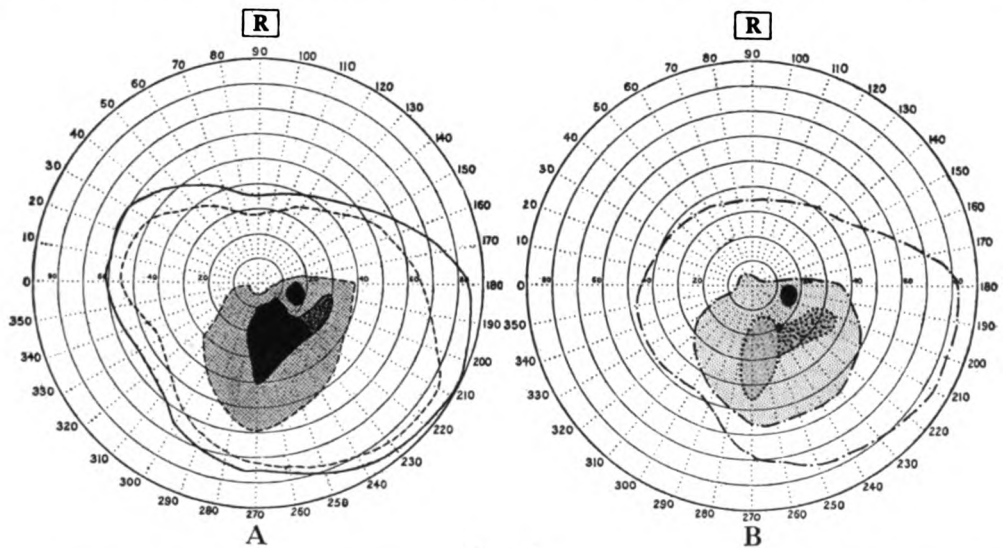


FIG. 42.

- A. Sarcoma of Choroid, with great elevation at site of tumour and deep fluid detachment above, coming down to and elevating macular area.
 Periphery for $\frac{3}{30}$ and $\frac{3}{30}$ normal. Defect for $\frac{3}{30}$ white does not include fixation area.
 B. Isopters for red and blue $\frac{3}{30}$ from same case. Peripheral boundary for red omitted. The defect for blue is larger than that for red and includes fixation area. Red object dimly seen in dotted area.
 Blue - - - - - Red V = $\frac{6}{60}$, excentric. (D. 1920.)

addition to the solid elevation of the retina, an elevation by fluid is produced. As Parsons has pointed out, this detachment often comes on early while the tumour is quite small and, especially if the tumour is in the upper hemisphere of the eye, may be quite separate from it. There may or may not be a detachment at the site of the tumour, and when present it may be relatively small.

The field changes are firstly those due to interference with the retina by the tumour, and, secondly, those due to the detachment. The relative prominence of the two sets of changes depends upon the stage at which the detachment occurs, and the area of retina which is in contact with the tumour.

A scotoma develops corresponding to the site of greatest damage to the retina. It might be supposed that, as the photo-chemical elements suffer first, such a scotoma would be characterised by relative blue blindness, but, in a personally observed case, specially suitable for the examination of this point, the defect for blue was smaller than that for red, and both colours were well seen over the edge of the tumour. Visual acuity

does not appear to suffer severely at first, at any rate, on account of the elevation of the retina by a choroidal growth.

As the retina becomes damaged the scotoma becomes more intense at the injured part, and when the tumour emerges into the vitreous it appears rather to push the nerve fibres apart than to destroy them. Were the fibres interrupted which pass over the site of the tumour the field defect would include a peripheral area corresponding to the part of the retina from which the fibres are derived. Roenne (352) points out that this does not occur, and in my case the field area concerned was only slightly depressed. It would, therefore, appear that the retinal fibres, like those of other parts of the visual path, are resistant, and can suffer much deformation and stretching before becoming functionally impaired.

When detachment of the retina becomes superadded, the signs already described are present, and in the case mentioned interlacing of the fields for red and blue was the first sign elicited. The field now shows the usual perimetric features of detachment, together with, at one part, a more intense scotomatous area corresponding to the most damaged part of the retina. According to Berry (33) and Domec, detachment due to tumour is characterised by sharp definition of the field defect, and the latter observer also noted that the extension found on examination in dim light was less generally present in tumour cases. In simple detachment, central vision is nearly always more or less reduced, in tumour cases it is more often good or only moderately impaired, unless the macular area is directly affected. The conditions found will naturally vary with the circumstances of each case. Should the detachment come on early, it is possible that, as Parsons has shown, the scotoma corresponding to the tumour may be much less intense than the defect over the detachment. It is also probable that a peripheral defect due to interference with the nerve fibre layer by the growth of the tumour will not be frequently observed as the detachment may become very extensive or other changes may supervene, or the eye be removed, before this stage is attained.

As regards the practical problem of diagnosis, it is evident that, in cases of recent detachment of doubtful causation, the presence of a circumscribed more intense scotoma within the relative defect, or of two separate defects one above and one below, is suggestive of tumour, and indicates examination of the quality of the changes. It is perhaps unlikely that perimetry will be frequently required in the diagnosis of choroidal tumour, but as a supplementary method, especially in the early stages and in doubtful cases, it is deserving of attention, while, in addition, it may yield results from the point of view of research.

Other Affections of the Retinal Outer Layers

In thrombosis of the retinal vein Foster Moore found moderate contraction of the field with occasional irregular defects and a practically constant central or paracentral scotoma which may be relative and may require a dim light for demonstration. Where a branch only was affected, no sector defect corresponding to the drainage area of the vein was observed, and no central scotoma unless the vein was directly concerned with the macular area. Moore's observations are of interest as indicating that the venous back

pressure and hæmorrhages in the inner retinal layers do not seriously affect the conducting power of the fibres, in contradistinction to the results of similar lesions in other parts of the nerve path where the fibres are more confined. The scotoma may be explained by the œdema and subsequent changes affecting the outer layers of the central part of the retina found by Williamson-Noble.

According to Köllner, acquired relative blue blindness in the macular area is frequently present in commencing albuminuric retinitis. It may also be elicited in macular œdema from any cause, with or without star figure. In cases of high intracranial pressure with gross swelling of the optic disc the detection of a relative central

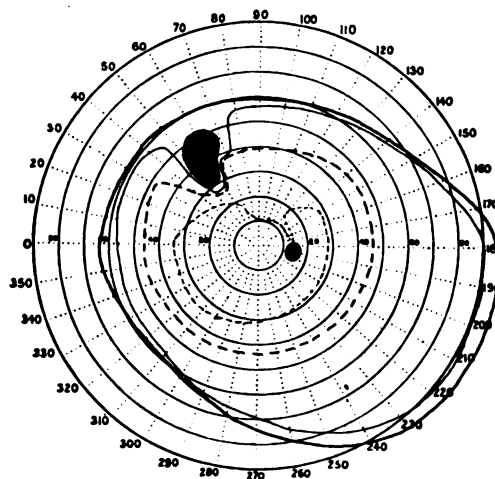


FIG. 43.—ANGIOMA OF RETINA.

Field of right eye, showing peripheral scotoma corresponding to the angioma which was in the lower outer quadrant of the retina. The defect in the field for $\text{I}^{\text{st}} \text{V. §.}$ is due to the large blood vessels. It is seen to be directed towards the peripheral scotoma. Objects $\text{S}^{\text{st}} \text{V. §.}$, $\text{S}^{\text{st}} \text{V. §.}$, $\text{I}^{\text{st}} \text{V. §.}$, $\text{I}^{\text{st}} \text{V. §.}$. V. §. (M. N. 1930.)

scotoma of this type, presenting retinal outer layer characters, is of value in the diagnosis of the cause of the visual failure and in preventing confusion with other causes such as optic atrophy or local pressure on the nerve path.

II. CONDITIONS AFFECTING THE CONDUCTION APPARATUS IN THE RETINA

Apart from the toxic amblyopias and glaucoma the retinal part of the conducting apparatus suffers in certain choroidal and vascular lesions, and also in injuries, which interrupt the functional continuity of the nerve fibres. Such interference produces field defects of nerve fibre bundle type whose distinctive characters have already been described.

Injury

Injury of the inner layer of the retina by a penetrating foreign body may cause a nerve fibre bundle defect. (Dalsgaard-Nielson 75, Groes Petersen 147). This is a simple form of nerve fibre bundle defect comparable to a laboratory experiment.

Acute Exudative Choroiditis

As has been pointed out, the nerve fibre layer is not affected by the lesions of ordinary disseminated choroiditis. On the other hand, certain acute exudative inflammatory foci, which differ from the disseminated type in that they are few in number or solitary, often penetrate to the fibre layer. The visual consequences reflect the amount of damage done and consist of a field defect corresponding to the injury to the rods and cones, together with, in proportion to the depth to which the lesion has penetrated the retina, a second defect corresponding to the area of distribution of the nerve fibres involved. In mild cases the outer layer defect may be present alone ; when the condition is more severe the inner layer defect is added.

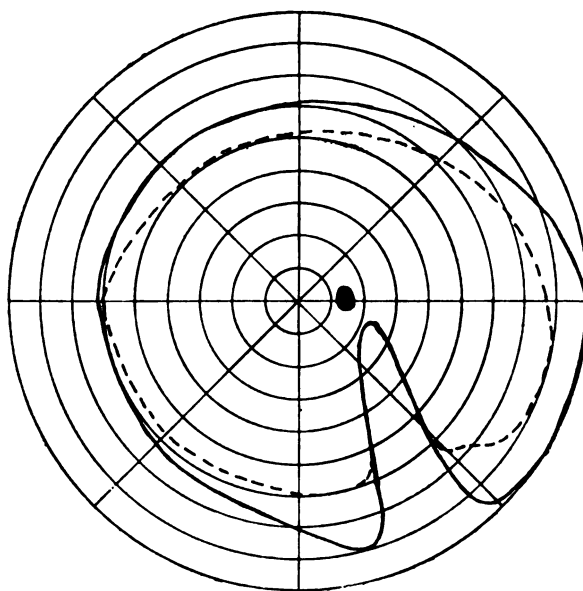


FIG. 44.—INJURY OF RETINA.

Nerve fibre bundle defect in lower temporal quadrant due to a foreign body which has lodged in the retina above the optic disc. (Dalsgaard-Nielson 75). Courtesy of the editor and publisher of the *Trans. Ophth. Soc. U. K.*

A lesion of this kind occurring at the edge of the optic disc is known as choroiditis or retino-choroiditis juxta-papillaris (Jensen), and produces the characteristic arcuate or cuneate nerve fibre bundle defect. When the lesion is at a distance from the disc the defect is of the same nature, but truncated (Fig. 45A). The scotoma corresponds to the retinal area from which the affected fibres come, and nearly always extends to the periphery. As is so common in nerve fibre bundle defects, the fixation area is unaffected unless the lesion directly affects the macula or the temporal side of the papilla. The scotomata may occur in any part of the field, on the temporal as well as on the nasal side, so that both cuneate and arcuate types of nerve fibre bundle defect are well represented, in contradistinction to glaucoma in which cuneate defects are rare. The lower part of the upper lateral quadrant is said to be seldom affected. The scotomata are, as a rule, sector shaped and comparatively wide and extensive occupying from half to one

and a half quadrants of the field or more. More rarely they are elongated and narrow. When arcuate they resemble the Bjerrum defect of glaucoma when it is well advanced, but they rarely imitate the narrow paracentral sharply curved defect found in the central field in glaucoma. The shape and position of the defects can be correlated by ophthalmoscopic examination with the position of the visible lesions, bearing in mind the course of the nerve fibres in the retina. The defect always extends to the position of the lesion from which it is never separated, as may occur in glaucoma, by an area of unaffected field. Defects at a distance from the blind spot are due to lesions correspondingly situated. The size of the field defect does not necessarily correspond to the size of the visible lesion, as the appearance of the lesion does not indicate exactly how much damage has been done to the fibre layer. The intensity is nearly always absolute and

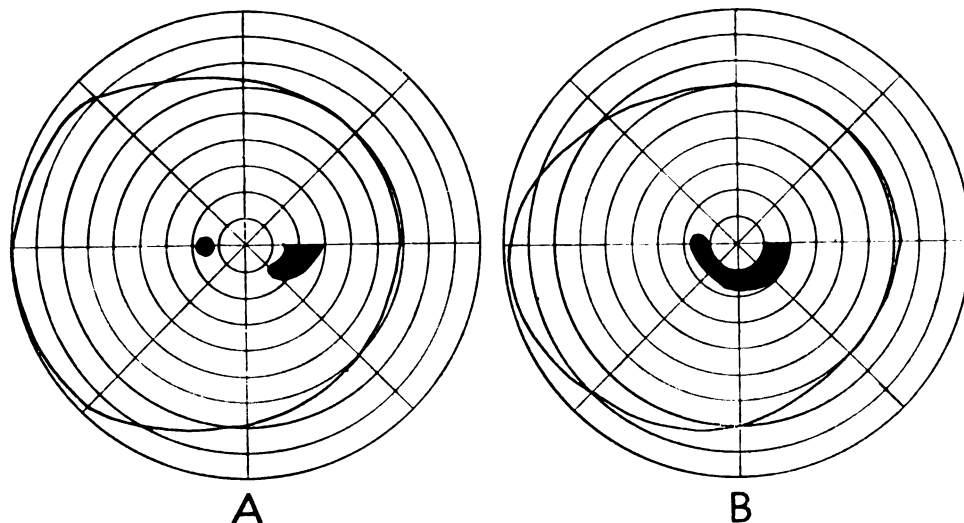


FIG. 45.—CHOROIDITIS JUXTA-PAPILLARIS.

Nerve fibre bundle defects. (Groes-Petersen.)

In A the lesion has been at a little distance from the optic disc.

Courtesy of the editor and publisher of the *Trans. Ophth. Soc. U. K.*, 1944.

uniform, rarely relative, and the edges are steep, but if the fields are tested while the choroidal lesion is still active, relative defects with disproportion for colour may be found. At this stage, however, the findings may be modified by the presence of vitreous opacities.

Interpretation.—As the defects agree very closely with the nerve fibre distribution it is evident that they are due to interference with the fibres at the site of the lesion. The lesion causes a defect corresponding to its extent and depth, and in addition, a radiating nerve fibre defect which represents the retinal area from which the damaged nerve fibres come. When there are two radiating defects, as in Fig. 48, it may be supposed that the destructive effect of the lesion has not penetrated the nerve fibre layer equally so that the fibres corresponding to the isolated part of the field have escaped. A similar interpretation may be applied to the field changes shown in Fig. 49 in which the upper absolute defect does not extend to the blind spot while the lower one does

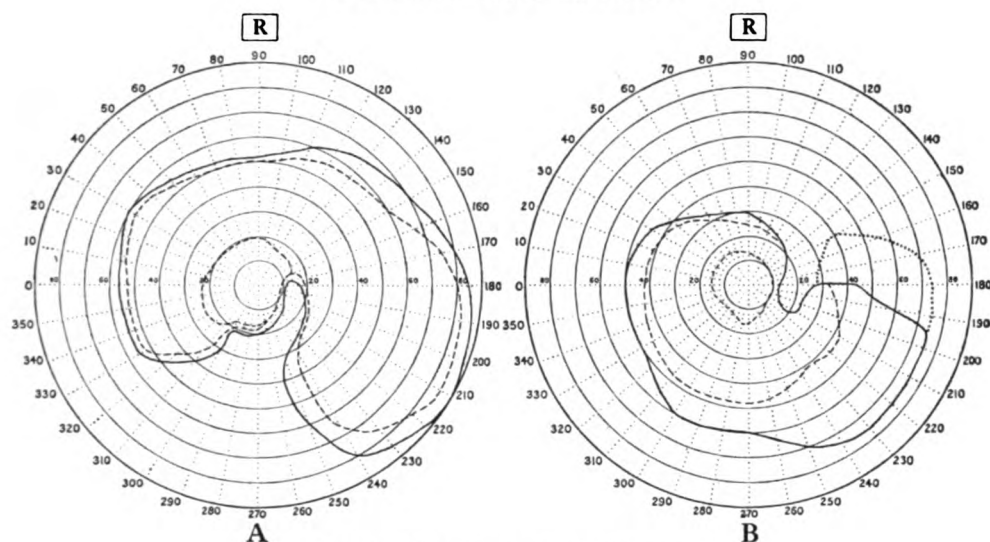


FIG. 46.—CHOROIDITIS JUXTA-PAPILLARIS.

Arcuate scotoma involving blind spot.

- A. A. M., age 22. $\frac{3}{3} \frac{3}{0} : \frac{3}{3} \frac{3}{0} : \frac{1}{2} \frac{0}{0} \frac{0}{0} \frac{0}{0} \frac{0}{0} \frac{0}{0}$.
 B. L. I., age 31. $\frac{3}{3} \frac{3}{0} : \frac{1}{1} \frac{0}{0} \frac{0}{0} \frac{0}{0} \frac{0}{0} \frac{0}{0}$. $\frac{3}{3} \frac{3}{0}$ dimly seen in area surrounded by dotted line.

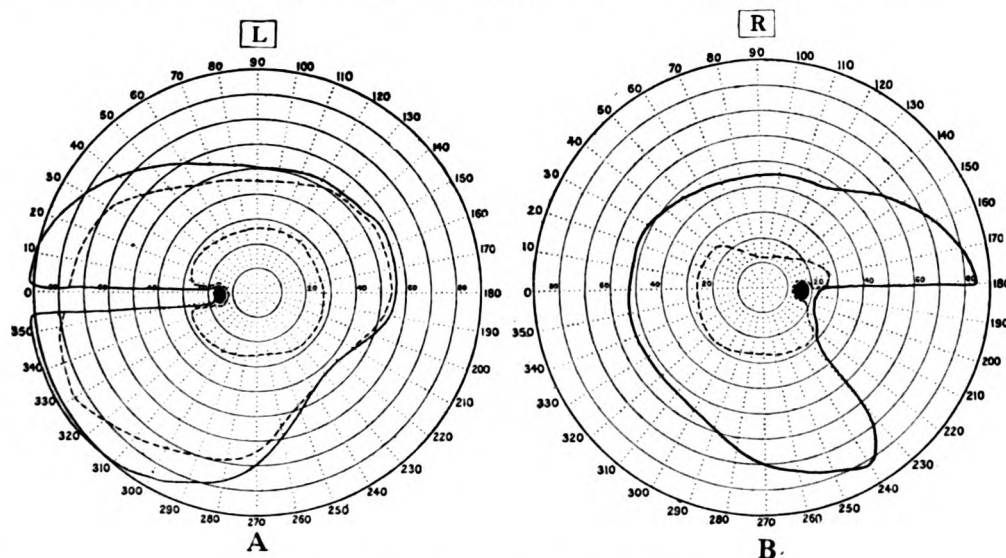


FIG. 47.—CHOROIDITIS JUXTA-PAPILLARIS.

- A. Horizontal radiate scotoma on temporal side of blind spot corresponding to lesion on medial side of optic disc. Objects $\frac{3}{3} \frac{3}{0} : \frac{3}{3} \frac{3}{0} : \frac{2}{2} \frac{0}{0} \frac{0}{0} \frac{0}{0} \frac{0}{0} \frac{0}{0}$. V = $\frac{1}{2}$ +. (L. F., age 19, 1925.)
 B. Wedge-shaped defect in temporal field corresponding to lesion at upper medial side of optic disc. The lesion was situated at a little distance from the disc, explaining the gap between the absolute defect and the blind spot. Objects $\frac{3}{3} \frac{3}{0} : \frac{2}{2} \frac{0}{0} \frac{0}{0} \frac{0}{0} \frac{0}{0} \frac{0}{0}$. (A. J., age 20, 1926.)

not extend to the periphery. It is possible that the damage may involve the retina in varying degrees, killing all or some of the fibres and producing relative or absolute defects accordingly. Careful examination with a wide range of quantitative tests is required to elucidate the conditions present and might possibly throw some light upon the layering of the fibres in the retina. The effect on the fibres is probably produced by

the occlusion of nutrient vascular twigs by the inflammatory focus or by cicatricial tissue. According to Uthoff (430) a small artery may become blocked.

Diagnosis.—The recognition of the field changes is of value in recent cases in distin-

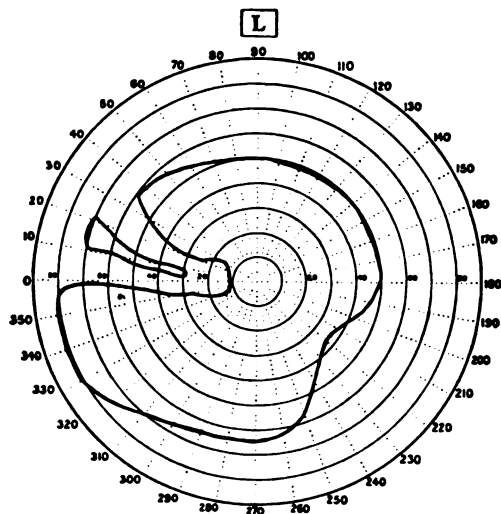


FIG. 48.—CHOROIDITIS JUXTA-PAPILLARIS.
Lesion on nasal side of papilla. Showing two temporal nerve fibre bundle defects separated by a narrow interval. V = $\frac{5}{16}$. S. 1937.)

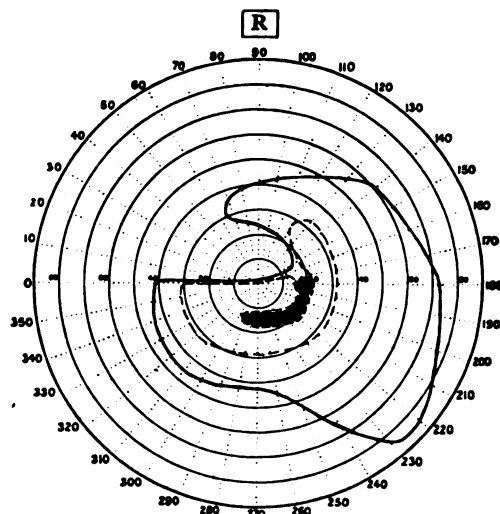


FIG. 49.—CHOROIDITIS JUXTA-PAPILLARIS.
Nerve fibre bundle defects above and below blind spot. Depression of field. V = $\frac{5}{16}$. Objects $\frac{1}{350}$, $\frac{2}{280}$, $\frac{3}{200}$. (M. S. 1937.)

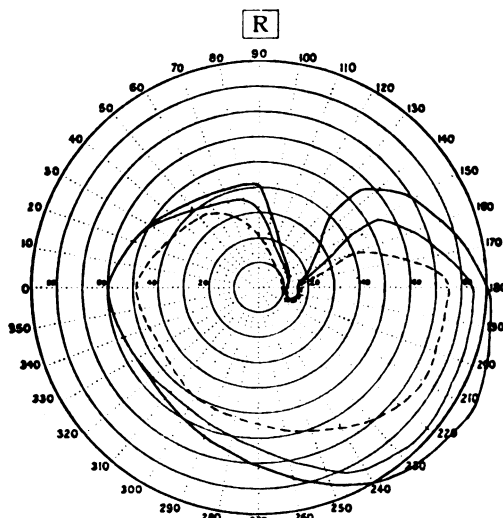


FIG. 50.—CHOROIDITIS JUXTA-PAPILLARIS.

Field taken before active stage has subsided showing sloping edge of defect. V. $\frac{5}{16}$. (G., 1930.)

guishing the condition from optic neuritis, which it may resemble owing to the prominent swelling of the disc, and in older cases from post-neuritic atrophy. In shape the field changes resemble other nerve fibre bundle defects such as those of glaucoma, but no mistake can possibly be made if all the characters are considered.

Prognosis.—The fields in these cases are usually examined when the active stage

has subsided, and the absolute, uniform and steep-edged defects then found are stationary and permanent. In the earlier stages the defects which are present may diminish, and even disappear, as the disease subsides provided that the damage done to the fibres has

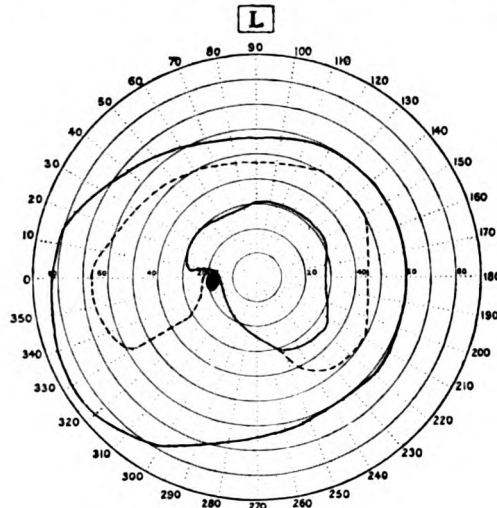


FIG. 51.—ARTERIAL DISEASE.

Relative defect extending to blind spot. Vision $\frac{5}{6}$ plus. Test-objects $\frac{5}{330}$, $\frac{1}{330}$, $\frac{1}{2000}$ (A., 1919.)

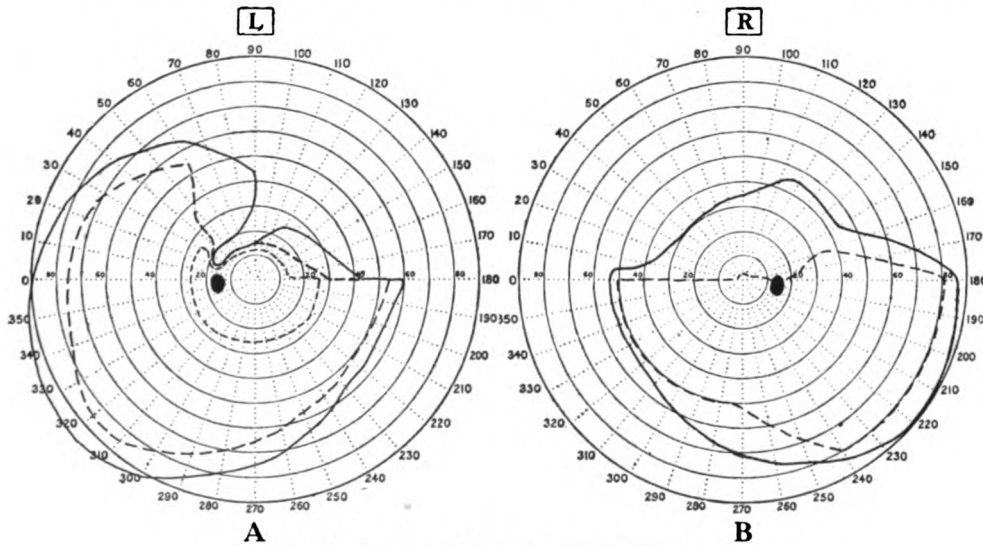


FIG. 52.—ARTERIAL OBSTRUCTION (ANGIOSPASM).

A. Miss I—æ. 25 years (1923). Third attack. V = $\frac{5}{6}$. Objects $\frac{5}{330}$, $\frac{1}{330}$. Central field $\frac{1}{2000}$.
 B. Miss E—æ. 21 years (1918). First of two attacks. V = $\frac{5}{6}$. Objects $\frac{5}{330}$, $\frac{1}{330}$.
 Note general similarity to field changes in glaucoma.

not been too severe. The prognosis in respect of central vision is good unless the macula is directly affected (Heath 161).

Obstruction of the Retinal Artery

Angiospasm, embolism or other forms of obstruction may affect the retinal artery or one of its branches, causing ischæmia of the nerve fibre and ganglion cell layers.

Both eyes may be affected at intervals; simultaneous bilateral retinal spasm may occur but is extremely rare. Many cases commonly regarded as embolism are really examples of spasm. The most interesting cases of spasm are those which affect young and otherwise healthy individuals.

The main trunk or a branch only may be affected. A cilio-retinal twig or a small branch arising above the lesion and remaining patent may be responsible for a spared area. In complete obstruction of the trunk of the central artery, Coats found a retained area of the field near the blind spot, extending temporally to a greater extent than nasally, suggesting a ciliary source of blood supply to the circumpapillary retina.

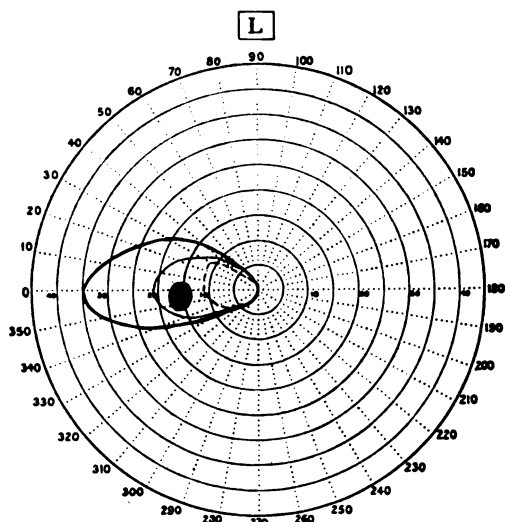


FIG. 53.—ARTERIAL OBSTRUCTION.

Complete loss of field with exception of the centro-caecal area. The papillomacular area of the retina showed two unaffected cilio-retinal arteries. Objects $\frac{4}{330}$, $\frac{3}{330}$, $\frac{1}{2000}$. V. $\frac{4}{6}$. (J. S., 1929.)

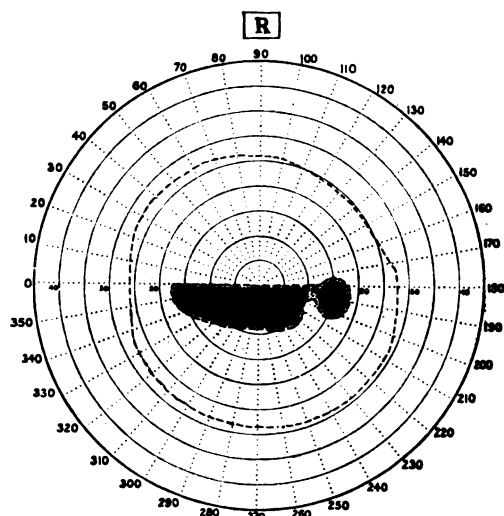


FIG. 54.—ARTERIAL OBSTRUCTION.

Horizontal hemianopic defect with division of fixation area. V. = $\frac{4}{6}$. Objects $\frac{2}{330}$ (scotoma and blind spot) and $\frac{2}{2000}$ (interrupted line). (A. B., 1923.)

In complete obstruction the patient usually gives a history of sudden and total blindness but on testing by the confrontation method with the hand or a large white object some vision will be obtained in the blind spot region. In cases of sudden blindness of one eye the correct diagnosis can often be made in this way.

The defect due to branch obstruction is of the nerve fibre bundle type, and is sector-shaped, having its apex at or near the blind spot. The sector is usually fairly large or may occupy about half of the field according to the distribution of the affected branch. Usually the upper or the lower half of the field is affected. The centrocaecal area, if separately supplied, may be preserved alone, or, if its supply only is obstructed, may show the only defect—a rare condition. De Schweinitz and Holloway found that a large central scotoma may be an onset symptom, a small central scotoma remaining permanently, a rare occurrence which they attributed to an affection of the macular bundle due to oedema of the nerve caused by the arterial obstruction. Sometimes a very large area of the field, inconsistent with the ophthalmoscopic appearances, may be affected at first, recovering to a sector defect in a short time. This

may be due to an interference with the capillary supply of the nerve fibres on the disc, restored subsequently by collateral circulation, or to a partial relaxation of the spasm.

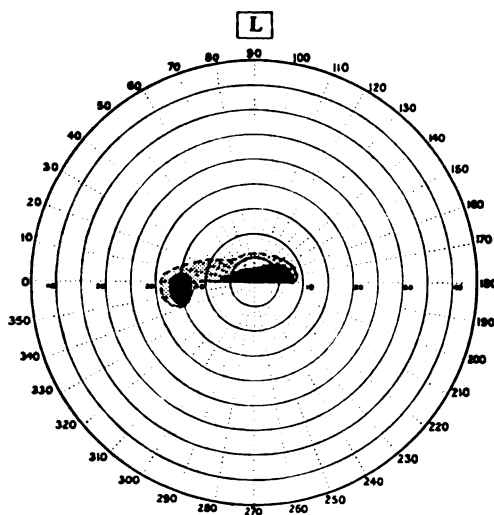


FIG. 55.

Female, age 66. Third attack of spasm. Field tested about a year later. Fixation area involved. Vision $\frac{1}{4}$. Objects: blind spot and scotoma $\frac{2}{200}$; relative scotoma $\frac{2}{200}$. (L., 1933.)

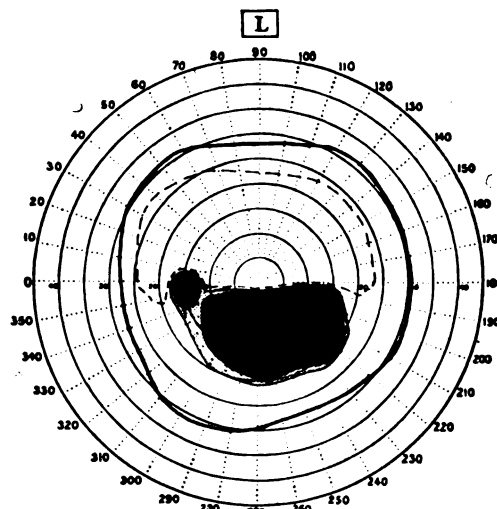


FIG. 56.

Male, age 23. Seven days after onset of spasm. Horizontal hemianopic defect sparing fixation area. Vision $\frac{1}{2}$. Objects $\frac{2}{200}$ (scotoma and blind spot). $\frac{2}{200}$ unbroken line, $\frac{2}{200}$ ---. (W. C., 1936.)

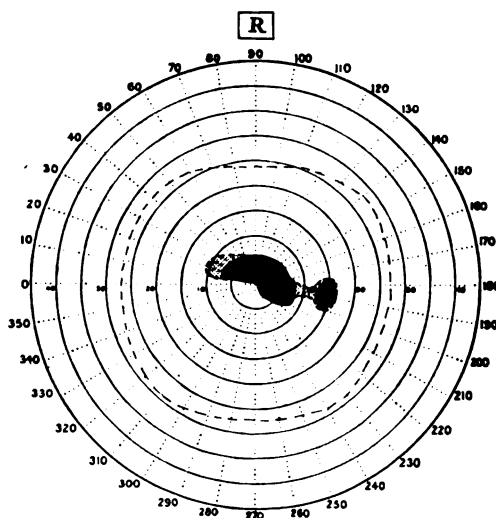


FIG. 57.

Male, age 55. Scotoma affecting central field both above and below horizontal meridian, but avoiding fixation area. Occurred after symptoms of migraine and vascular disturbance, elsewhere as well as ocular, for 19 years. Vision $\frac{1}{2}$. Objects $\frac{2}{200}$ blind spot and scotoma, $\frac{2}{200}$ relative scotoma and field outline. (McM., 1932.)

Partial obstruction commonly takes the form of occlusion of a temporal branch. The obstruction is usually situated at or near the disc and near a bifurcation of the

C.P.

1

artery. An upper or lower nasal sector or arcuate defect, with a straight boundary along the horizontal meridian, resembling the field change of glaucoma, is produced. As in glaucoma, the fixation area may be spared, the scotoma arching around it, but frequently it is divided horizontally, vision remaining good in the retained portion, so that the upper or lower half of a letter of $\frac{6}{6}$ may be read (Roenne 331). This feature is very exceptional in glaucoma. A suggested explanation is that the fovea is horizontally traversed by the vascular raphé whereas the nerve fibre raphé extends only from its temporal side to the periphery. Also the macular fibres, which are sheltered in glaucoma, are only rarely protected by a cilio-retinal twig in obstruction.

The part of the scotoma immediately adjoining the blind spot is not so dense as that a little further off, no doubt because the corresponding retinal area is not solely nourished by the occluded branch but also by circumpapillary cilio-retinal twigs. This feature also

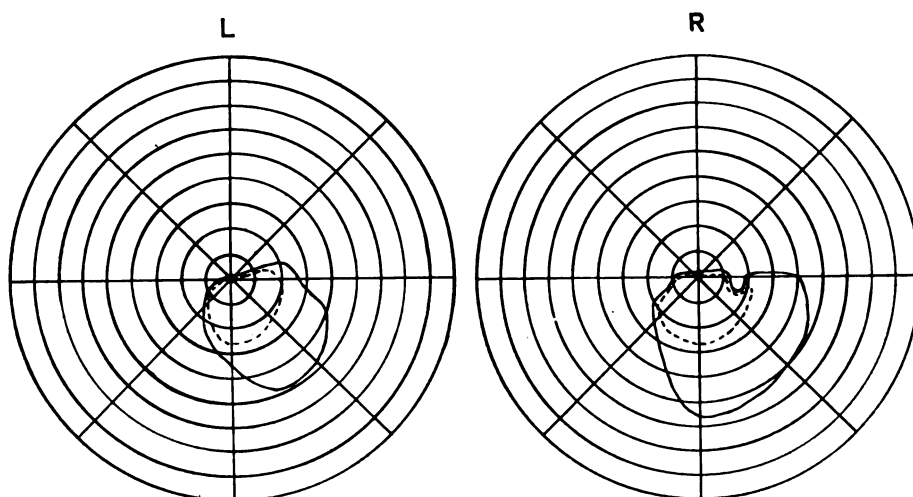


FIG. 58.—BILATERAL SIMULTANEOUS ARTERIAL OBSTRUCTION.
The macula has escaped in both eyes. Objects: $\frac{30}{30}$, $\frac{10}{10}$. V. R. and L. $\frac{6}{6}$. (S., 1947.)

occurs in the scotoma of glaucoma possibly, to some extent at any rate, for the same reason (p. 135).

It is noteworthy that branch obstructions are rarely found elsewhere than in the central part of the field. The arteries which arch over and under the macula appear to be specially liable to this form of disease. Those which pass to the nasal retina do not appear to be ever involved unless as part of an obstruction of the whole central artery or one of its two main divisions, and their smaller branches seem also immune. This cannot be explained by suggesting that defects due to such obstructions are missed because central vision is not affected. If such obstructions were at all common they would have been found in the ordinary course of ophthalmic examination. It would appear, therefore, that the vessels passing around the macular area are specially predisposed to the various forms of vascular obstruction.

The characters of the field changes indicate that the main cause of the defects is the loss of conducting power of the nerve fibres owing to interference with their blood

supply. The other factors present, such as œdema of the retina and ischæmia of the ganglion cells, have probably some influence but it is difficult to estimate its nature or amount. The watershed between the arterial branches arching over and those curving under the macular area is horizontal only in the neighbourhood of the macula (Fig. 257, p. 298), and is wavy or sinuous farther towards the temporal periphery of the retina. The field defects (Fig. 52) do not reproduce this outline but tend to resemble in shape the nerve fibre bundle defects of glaucoma. Should part of the affected area of the retina receive a fresh blood supply from adjacent sources this will not result in a diminution of the field defect unless the corresponding nerve fibres are also properly nourished throughout their whole length. An anastomotic supply to the macula cannot restore macular vision if the macular fibres pass through an ischæmic area at any point on their way to the papilla, and the exact results of the obstruction depend on the condition of the terminal arterial twigs near the papilla as well as at the macula.

The defects are usually absolute and steep-edged, and, once established, are permanent, indicating the practically complete loss of function within the affected area.

Diagnosis does not usually require the aid of perimetry, which is, nevertheless, of great value in old cases of branch obstruction when the history is vague and the ophthalmoscopic appearances inconclusive. When the case is seen some time after the occurrence of the obstruction the diagnosis is still easy and definite owing to the very characteristic features of the scotoma. The case shown in Fig. 55 was diagnosed about a year after its occurrence. Cases of spasm are recognisable by the history of repeated obscurations of vision before the production of a permanent defect. The scotoma in Fig. 57 occurred after frequent attacks over a period of nineteen years. The resemblance of the defect to that of glaucoma is likely to present difficulty only in certain cases of chronic retinal arterial disease with raised ocular tension sometimes met with in elderly persons. Even here thorough examination will always indicate the true nature of the case.

Nerve fibre bundle defects may also occur in retinal hæmorrhage if the bleeding has damaged the inner layer of the retina. A defect extending to the periphery may be produced in this way by a small hæmorrhage near the optic disc (Baas). Roenne (331) described a bundle defect in a case of albuminuric retinitis.

CHAPTER VIII

GLAUCOMA

No ocular disease is more intimately associated with perimetry than glaucoma. This connection has produced reciprocal advantages, for our clinical knowledge of glaucoma has been greatly advanced in recent years by perimetric studies, and it is largely to the investigation of the field of vision in this disease that perimetry itself owes its development.

Regarded from the aspect of the site of interference, glaucoma lies between affections of the retina and those of the nerve and while the chief interference is at the optic disc, there is also probably a certain amount of retinal impairment.

In the acute form with very high intraocular pressure, owing to corneal oedema and interference with the retinal arterial circulation the part played by perimetry is usually limited to a rapid and rough estimation of the area and acuity of whatever vision is present. The whole field is greatly depressed, sometimes predominantly on the nasal side if a chronic type of the disease has pre-existed.

In chronic glaucoma (glaucoma simplex) perimetry is of great importance, as it is by its means that the amount of damage done can best be ascertained and the rate of progress watched.

The main features of the well-developed glaucoma field are depression with peripheral contraction more pronounced on the nasal side, associated with a nasal step and, later, wide sector defects; the defects are relative at first, and absolute when well developed, and colour vision is not impaired in a disproportionate degree. Central vision is almost always retained until a late stage, when the peripheral field has mostly disappeared.

These features remained unexplained until the demonstration by Bjerrum of the nerve fibre bundle defect. The screen examination unclothed, as it were, and analysed the gross changes found by testing with large visual angles, and displayed their true nature and significance. The characteristic field changes of glaucoma are nerve fibre bundle defects, and in glaucoma this type of defect attains its highest development.

The first fibre groups to be affected are those which pass from the horizontal raphé some little distance beyond the temporal side of the macula around which they arch to enter the upper or lower, more often the lower, margin of the optic disc. The field defect corresponds to this anatomical arrangement and consists of a depression of the upper part of the central field between the blind spot and the horizontal meridian to the nasal side of the fixation point. As a rule this depression is most manifest near the blind spot, constituting the "baring of the blind spot" (Figs. 59, 61); sometimes it is apparent at an early stage on the nasal side in the form of a small "nasal step" (Fig. 63), in other cases both of these features may be present together (Fig. 62). Then a small curved scotoma appears lying between the 10° and 20° circles, usually in the upper temporal field quadrant, unconnected with the blind spot but so curved that, if continued, its temporal end would join the blind spot. In its earliest manifestation this scotoma can only

be detected by very small visual angles (*e.g.*, $\frac{1}{2000}$) and even then not always very definitely.

As it enlarges this defect extends towards the blind spot and arches nasalwards around the fixation area to the horizontal meridian of the field and the nasal periphery, becoming wider as it leaves the blind spot so that it comes to resemble a curved wedge or a comet. In this more developed stage it is known as the Bjerrum scotoma. The horizontal edge of the less affected or intact field on the nasal side of the fixation area corresponds to the horizontal raphé of the retina, and constitutes the "nasal step" of Roenne. As the intensity of the defect varies in different parts, usually greater peripherally, a series of test-objects is required to demonstrate its true extent.

Numerous intermediate forms occur, and as the changes are nearly always present both above and below the horizontal meridian, and always in different stages of development, an almost infinite variety of combinations may be evolved. Although the scotoma commonly begins in a temporal quadrant near the blind spot it may commence in a nasal quadrant, sometimes on the horizontal meridian, whence it gradually extends towards the blind spot. In some fields a well-developed scotoma extending to the blind spot is present on one side of the horizontal meridian while a scotoma extending from the raphé towards but not as far as the blind spot is present on the other side (Figs. 71, 75B).

If the examination is conducted with sufficient care it will be found that the curved defect is isolated and unconnected with the blind spot though occupying a curve which, if continued, would traverse it. In many well-developed examples with absolute defect, a narrow band of retained field may remain at the edge of the blind spot separating it from the scotoma. Even in advanced cases the caecal end of the arcuate scotoma may be less dense than the peripheral end. Thus extension of, or outgrowth from, the blind spot is not to be regarded as an initial sign of glaucoma, though a scotoma continuous with the blind spot is present in developed cases. The frequently repeated statement that the field changes in glaucoma simplex are ushered in by the appearance of tuft or flame-like defects extending from the poles of the blind spot originated in observations made with visual angles too large to elicit initial changes and have not been confirmed.

As has already been pointed out, the horizontal raphé may be regarded anatomically as a portion of the retinal periphery folded in to the fovea so that two portions of the periphery are here mutually opposed (Fig. 28). When the field area corresponding to one of these becomes depressed, the opposite area becomes demarcated by its horizontal straight boundary, corresponding to the raphé, which now forms the nasal step. The depressed area opposite the step may be traced towards the blind spot through the arcuate scotoma. The step feature is prominent when the demarcation takes place at the periphery of the field and when the exposed side is little affected (Fig. 70). If both upper and lower sides are approximately equally affected, which is unusual, there is little or no step. When an arcuate scotoma extends to the horizontal meridian between the fixation point and the nasal margin of the field, but not involving the latter, its curved sides and straight termination give it a shape somewhat resembling a Turkish scimitar with the handle at the blind spot (Fig. 78A). There may be a curved scotoma both above and below, and if complete and sufficiently symmetrical, these may together form a ring scotoma encircling

the fixation area and traversing the blind spot. A nasal step may be present in addition (Fig. 73).

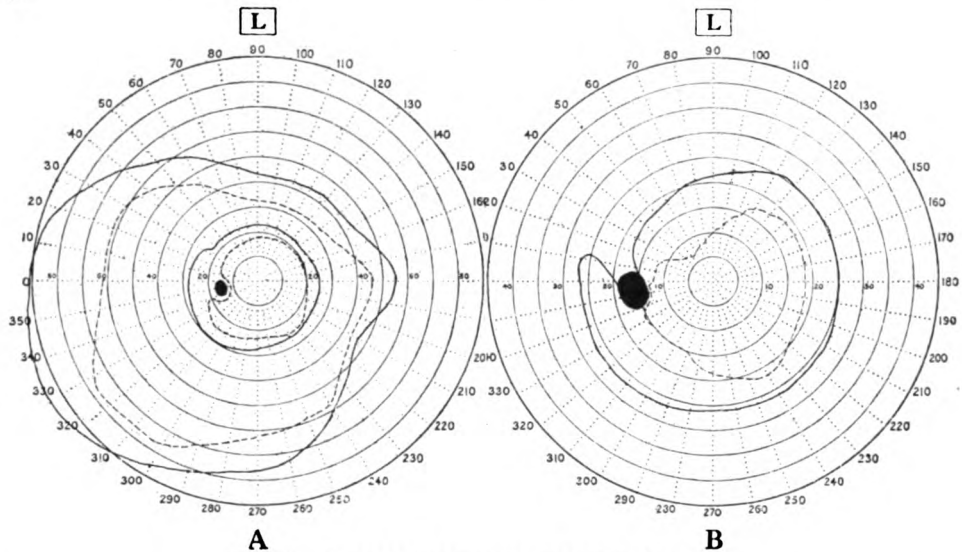


FIG. 59.—CENTRAL DEPRESSION IN EARLY GLAUCOMA.

- A. Isopters for $\frac{5}{30}$, $\frac{1}{30}$, $\frac{2}{30}$ and $\frac{1}{30}$, showing contraction of central isopters. Note baring of blind spot for $\frac{1}{30}$. Vision $\frac{6}{8}$ +. (M., 1924.)
- B. Another case. Central field only for $\frac{2}{30}$ and $\frac{1}{30}$. More definite alteration of central isopters. V. = $\frac{6}{13}$ +. Baring of blind spot for $\frac{1}{30}$ and $\frac{2}{30}$. Peripheral field normal. (J., 1923.) Acute glaucoma 1944.
- Both these eyes were glaucomatous, but without cupping or pallor of disc.

Sometimes the fibres on the temporal side of the optic disc become affected at an early stage, leading to a horizontal oval scotoma, composed of opposed small arcuate defects surrounding but sparing the fixation area (Fig. 79.L). Straight cuneate defects on

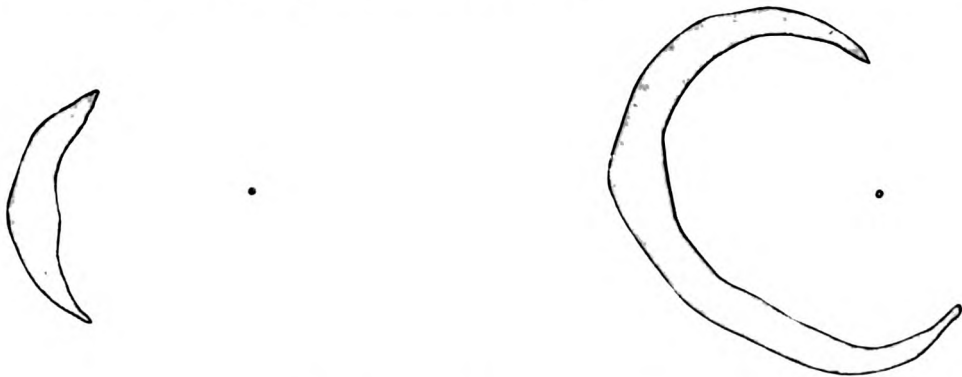


FIG. 60.—SEIDEL'S SCOTOMATA. $\frac{3}{100}$.
Seidel. (371.)

the temporal side of the blind spot seem to be extremely rare as initial field changes and are very uncommon even as late developments in the course of the field changes. The incidence of the bundle defects is least near the horizontal meridian and greatest towards the poles of the blind spot. The temporal field is lost partly by the lateral extension of

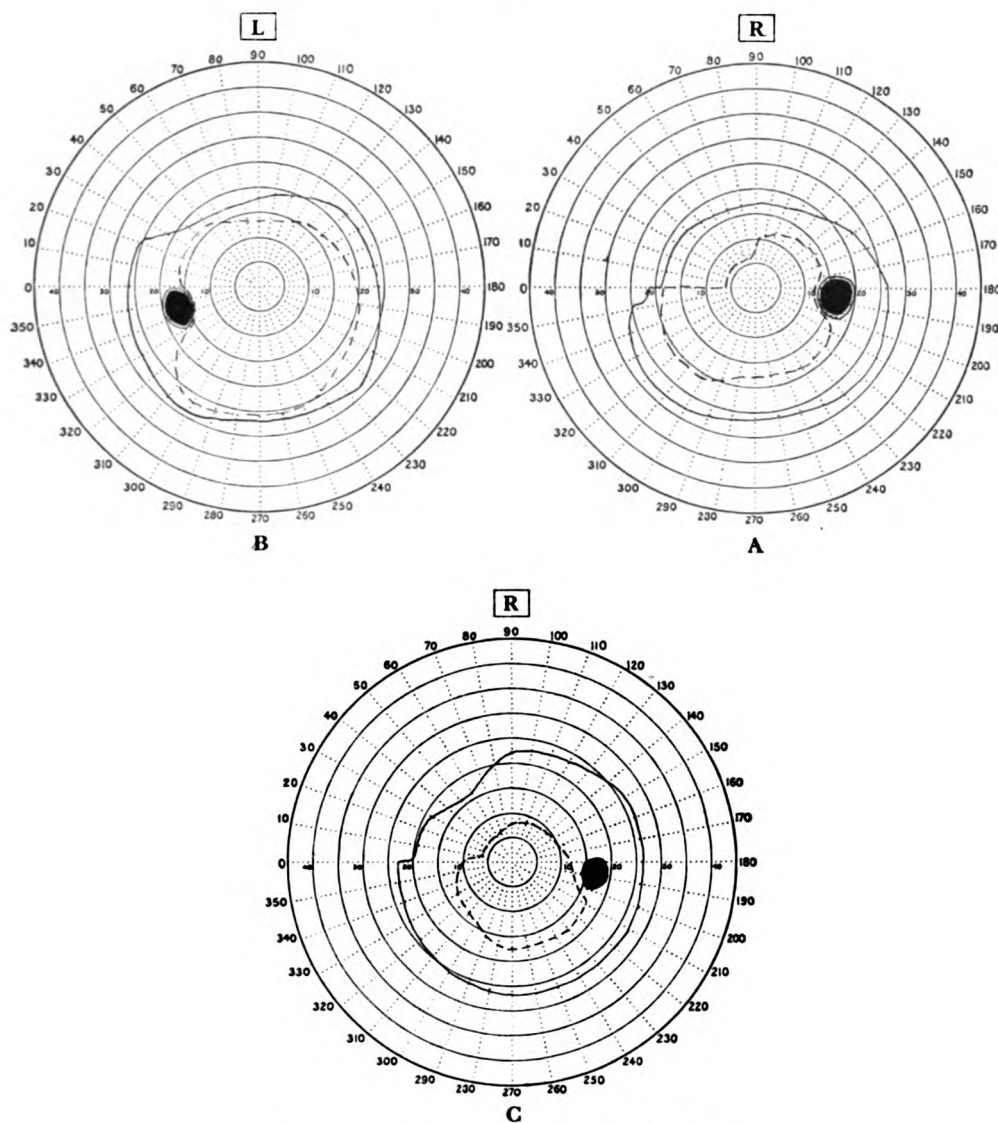


FIG. 61.—GLAUCOMA; EARLY FIELD CHANGES.

- A. Central field of right eye shows baring of blind spot, depression above and nasal step. Periphery normal. Disc cupped. T. 43 falling to 12 with pilocarpin. V. $\frac{5}{3}$. Objects $\frac{4}{000}$, $\frac{2}{000}$, $\frac{3}{000}$.
 B. Left central field shows commencing upper depression with baring of blind spot for $\frac{2}{000}$. Periphery normal. V. $\frac{5}{3}$. T. 19. Disc normal. Objects as for right eye.
 C. Right central field of same case after seven months pilocarpin. Increasing central depression. Periphery normal. V. $\frac{5}{3}$ +. T. 13. Disc shows no cupping. Objects as before.
 Left central field at this time unchanged but tension rising. (W., 1932.)

defects which have originated near the poles and partly by depression, the mode of loss differing greatly from that which takes place in the nasal field.

Peripheral isolated scotomata have been reported by Simon (374), but are probably not a real part of the perimetric picture of glaucoma.

In addition to the characteristic nerve fibre bundle defects peripheral and general depression also occur in greater or lesser degree, more pronounced in some cases, and

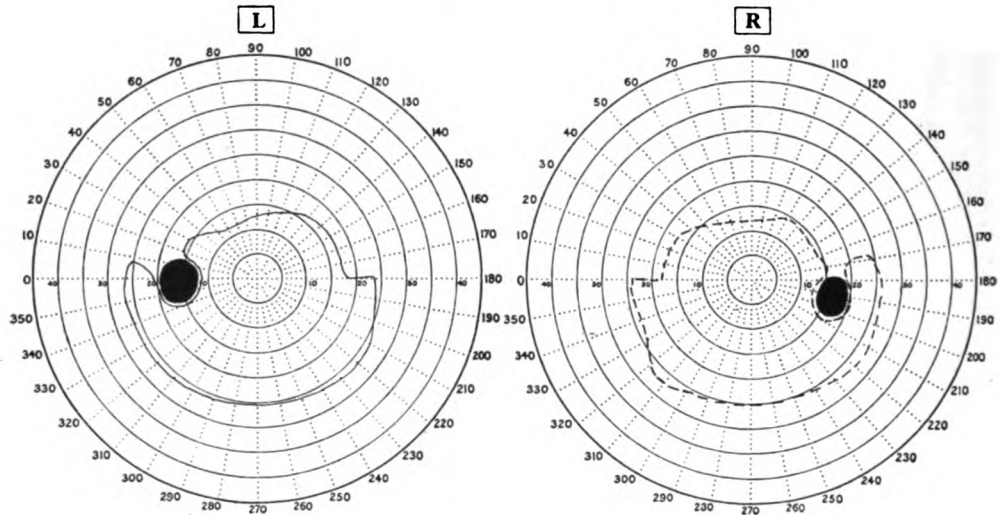


FIG. 62.—EARLY FIELD CHANGES IN GLAUCOMA.

History of halos for ten years in both eyes.

R.E. V. 8. T. 37. Disc normal. Central field shows baring of blind spot, depression of upper central field, and nasal step for $\frac{1}{2}$ of field.

L.E. V. 8. T. 56. Disc cupped. Central field similar to that of right eye but object $\frac{2}{3}$ of field. Peripheral fields normal. (C., 1933.)

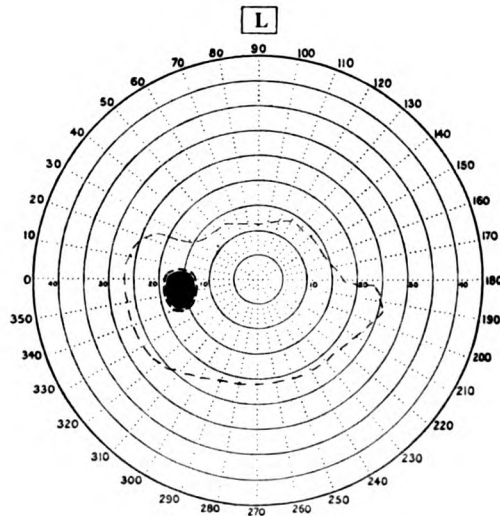


FIG. 63.—EARLY GLAUCOMA.

Depression of upper central field of left eye with nasal step but without baring of blind spot. V. 8. T. 28. Disc cupped. Peripheral field showed only a slight indication of a small step in the upper nasal quadrant. Right eye, absolute glaucoma. (W., 1926.)

in some parts of the field than in others. It is manifested by contraction of the internal isopters in the less affected, or apparently unaffected, areas, and may not be evident in the temporal field until the changes on the nasal side are well advanced. A noteworthy feature is the strong tendency to symmetry of the defects when both eyes are involved. Thus symmetrical sectors may be lost, or both upper or both lower halves of the fields.

When well developed these field changes can be easily elicited by the perimeter,

at least as regards their denser parts, but, in their early stages or when of low intensity, the screen is essential. It is also much easier to use, and more accurate than the perimeter, especially in regard to the important changes which lie within the 30° circle. The intensity of the defects is always graded, so that several test-objects are required to elicit the full extent and degree of the visual loss. What to a large object appears merely as a nasal restriction of the field may be shown in this way to extend almost as far as the blind spot and one of the commonest errors in the perimetry of glaucoma is the failure to trace the connection between the nasal defect and the blind spot region.

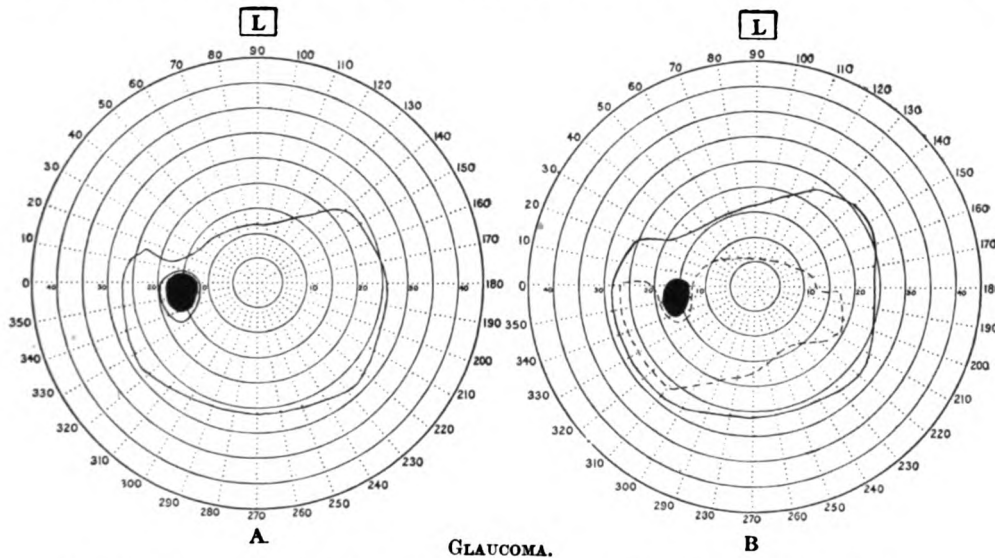


FIG. 64.—CENTRAL FIELD OF LEFT EYE OF SAME CASE AS FIG. 65 FIVE YEARS LATER.

A. Commencing depression above and threatened baring of blind spot. Periphery normal. $\frac{2}{3} \frac{0}{0} \frac{0}{0}$ blind spot, $\frac{2}{3} \frac{0}{0} \frac{0}{0}$. V. $\frac{6}{6}$. T. 35. Disc normal. (Ths., 1931.)

B. Same field two years later. Eserin and pilocarpin constantly used: T. 21. Disc normal. V. $\frac{6}{6}$. Depression of upper central field with baring of blind spot and nasal step. Objects $\frac{4}{3} \frac{0}{0} \frac{0}{0}$ blind spot, $\frac{2}{3} \frac{0}{0} \frac{0}{0}$, $\frac{2}{3} \frac{0}{0} \frac{0}{0}$. (Ths., 1933.)

In 1935 conditions similar but aggravated. Periphery normal.

In 1941 similar outlines were obtained but with larger objects.

Areas of doubtful or uncertain vision for a particular size of object and of rapid exhaustion are common. In advanced chronic cases the defects are often absolute with steep edges. As in all conduction defects colour vision is lost before that for white but there is little of the pronounced disproportion present in progressive optic atrophy such as that of tabes. As a rule the changes first involve the central field, surrounding but not invading the fixation area, and usually soon afterwards the periphery, but sometimes the central changes are already serious before the periphery is demonstrably affected.

We may now examine the relationship of the field changes to the onset and course of the disease.

At first, the rise of tension is transitory, and the subjective symptoms vary accordingly. Early field changes may be found when the tension is raised, and may be undemonstrable when it has fallen, whether spontaneously or following treatment. The field may be normal even when the tension is raised. In other cases there is slight

depression, sometimes confined to, or, at any rate, most easily elicited in the central field. This is shown by contraction of the internal isopters, so that, for example, the isopter for $\frac{1}{2000}$ passes to the nasal side of the blind spot, instead of surrounding it. Sometimes the isopter for $\frac{1}{2000}$ is contracted, mainly, or only, above, and normally situated below encircling the lower, but not the upper, part of the blind spot, so that the upper or upper outer part of the blind spot is exposed or bared. It is rare to find this exposure at the lower pole of the blind spot. When the $\frac{1}{2000}$ isopter is much contracted the baring of the blind spot may be demonstrable with an object of twice the size— $\frac{2}{2000}$ (Fig. 59B). Sinclair (377) in 1906 and later Malbran (276) found the same feature in early cases. The depression is not constant, but disappears when the tension is lowered, and may be the

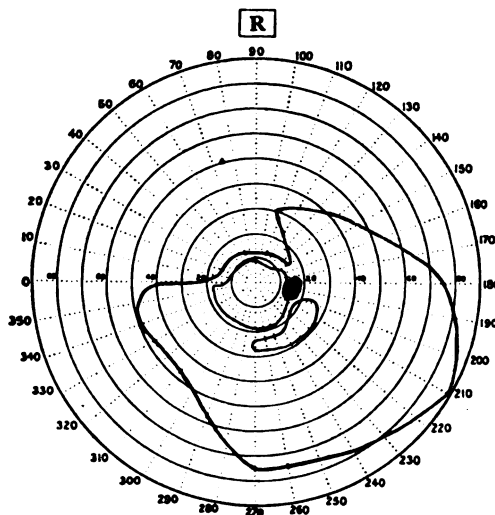


FIG. 65.—GLAUCOMA DISCOVERED DURING ROUTINE EXAMINATION.

Central field shows baring of blind spot, nasal step, and arcuate scotoma below for $\frac{2}{2000}$. Quadrant loss for $\frac{3}{3000}$ with arcuate scotoma not extending to blind spot. Objects $\frac{2}{2000}$ (blind spot), $\frac{2}{2000}$, and $\frac{3}{3000}$ periphery. Disc cupped. V. $\frac{5}{8}$ +. T. 43. (Ths., 1926.)
Field of right eye of patient whose left field, five years later, is shown in Fig. 64.

only perimetric sign in eyes definitely known to be glaucomatous. Taken by itself, it is, of course, not peculiar to glaucoma, though very suggestive when other indications are present.

The depression may extend across the upper part of the field, arching over the fixation area, to the horizontal meridian on the nasal side where it appears as a small nasal step. Occasionally the nasal step may be found without the baring of the blind spot, or both may occur together (Figs. 62, 63).

This depression increases slowly, and may become demonstrable all over the field though its intensity is slight. The arcuate defects may develop at a relatively early stage, when the depression is little in evidence, so that there may be advanced central defects with no perceptible peripheral field change, or the depression may be widespread though still slight in degree before they appear. They commence more frequently in the upper than in the lower field and apparently never simultaneously in both. In its early stages the arcuate scotoma is not in contact with the blind spot.

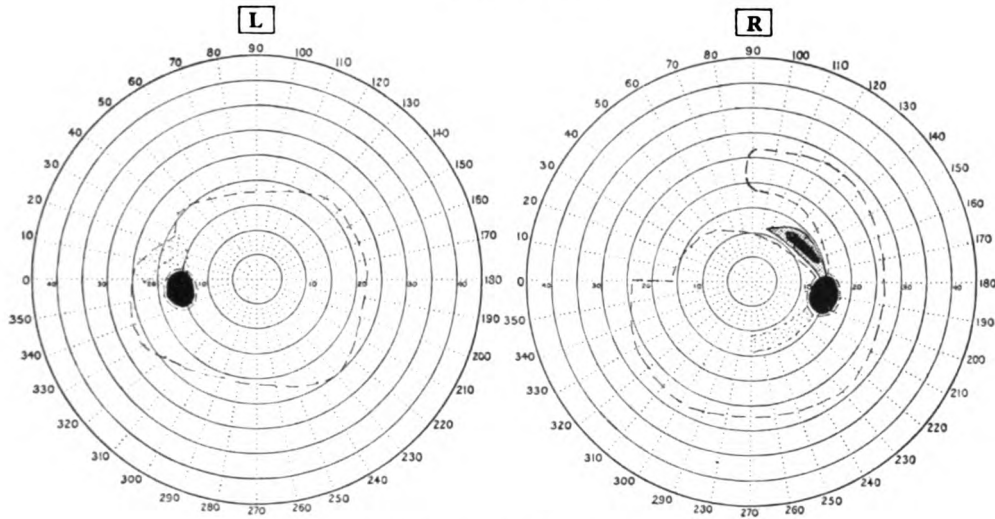
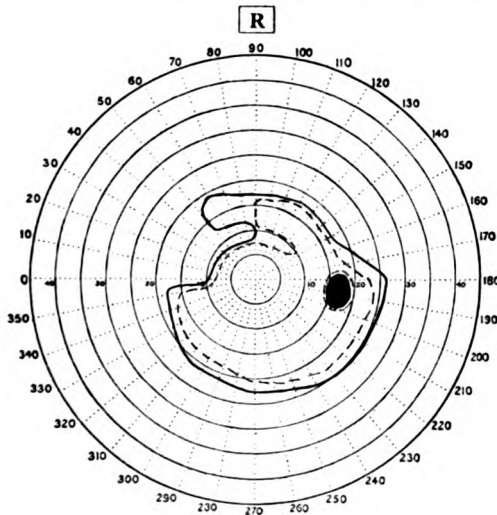


FIG. 66.—EARLY GLAUCOMA.

Central fields of a patient who had received several diagnoses both positive and negative. Vision $\frac{6}{6}$ plus and tension 15 in each eye. Both discs deeply cupped, especially right. Central field of right eye ($\frac{1}{2000}$) shows arcuate scotoma with nasal step. The dense part of the scotoma does not reach the blind spot. There is a very faint indefinite arcuate scotoma below for $\frac{1}{2000}$. Objects $\frac{1}{2000}$, $\frac{1}{2000}$. Left eye shows commencing depression of upper central field with a dim area upwards and temporally indicating threatened barring of blind spot. Objects $\frac{1}{2000}$, $\frac{1}{2000}$. Peripheral fields normal. (A., 1932.)



GLAUCOMA.

FIG. 67.—CENTRAL FIELD OF RIGHT EYE SHOWING ARCULATE SCOTOMA APPROACHING BLIND SPOT FROM HORIZONTAL MERIDIAN.

Note nasal step. Objects $\frac{1}{2000}$ blind spot, $\frac{1}{2000}$ and $\frac{1}{2000}$ (broken line). T. = 25. V. = $\frac{6}{6}$. Central cupping of disc. No pallor. (P., 1933.)

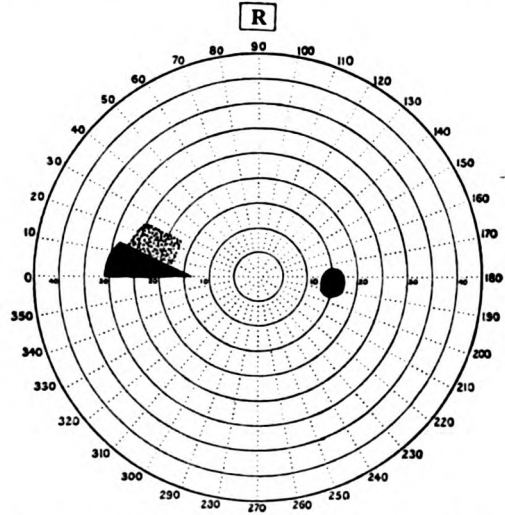
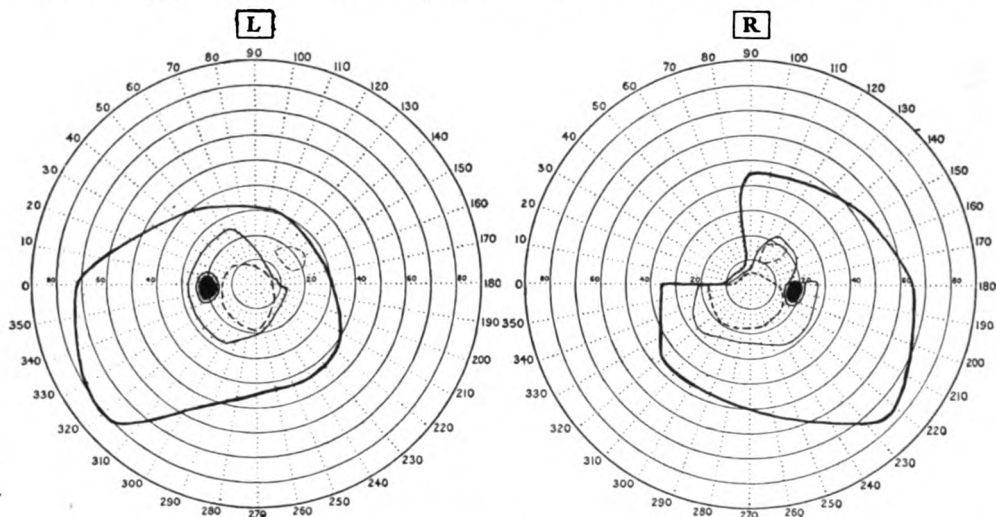


FIG. 68.—FROM A CASE OF GLAUCOMA SHOWING THE SCOTOMA ONLY ON THE NASAL SIDE.

Note the straight edge along the horizontal meridian. (Van der Hoeve, 441.)

It may be presumed that the earliest perimetric sign in glaucoma, or in any other gradually developing lesion of the visual path, is the sign which requires the weakest stimulus for its detection. The scotomata of Seidel were demonstrated with objects

of $\frac{3}{1000}$ and $\frac{5}{2000}$ corresponding to visual angles of $10\cdot3'$ and $8\cdot6'$ respectively, which are four or five times larger than are necessary to elicit relative defects of slight intensity. Using objects of $\frac{2}{1000}$ or $\frac{1}{1000}$, I have always found more extensive nerve fibre bundle defects,



GLAUCOMA.

FIG. 69.—FIELD CHANGES PRONOUNCED IN UPPER NASAL QUADRANTS; NASAL STEPS, BARING OF BLIND SPOT IN RIGHT FIELD, ABSENCE OF ARCULATE SCOTOMA IN NEIGHBOURHOOD OF BLIND SPOT.

R.E. Objects $\frac{7}{330}$, $\frac{4}{2000}$ (blind spot), $\frac{3}{2000}$, $\frac{1}{2000}$. V. $\frac{5}{6}$. T. 47. Disc cupped.
L.E. Objects $\frac{7}{330}$, $\frac{4}{2000}$ (blind spot), $\frac{3}{2000}$, $\frac{1}{2000}$. V. $\frac{5}{6}$. T. 42. Disc deeply cupped. (M., 1936.)

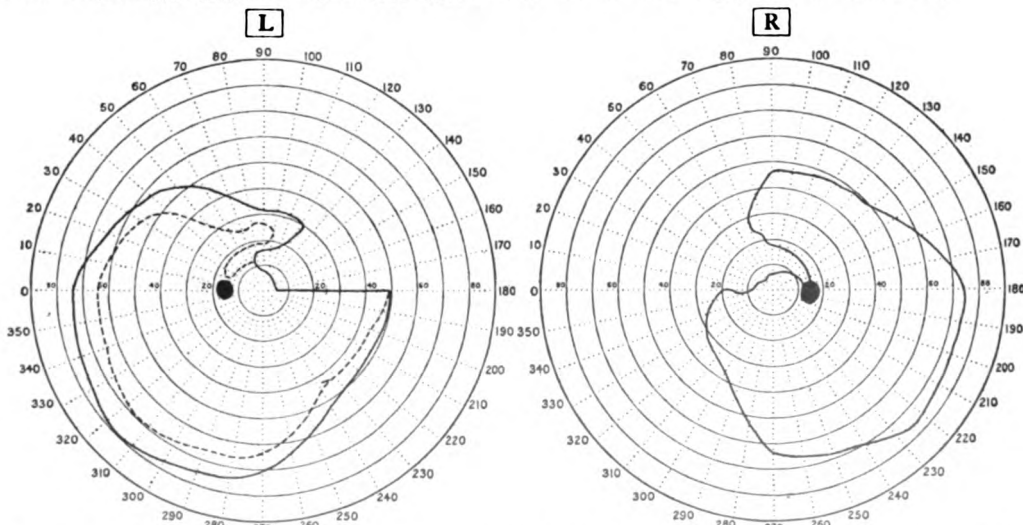


FIG. 70.—GLAUCOMA. FIELD CHANGES MODERATELY ADVANCED. NASAL STEP AND ARCULATE SCOTOMA.

V.R. and L. $\frac{5}{6}$.

In the left field the scotoma does not extend to the blind spot with the test-objects used, $\frac{3}{330}$ and $\frac{3}{330}$. (J. P., 1914.)

if any. This suggests that the Seidel scotoma may represent the more intense part of a larger relative defect. That the use of a delicate test should show that the arcuate type of scotoma in its inception springs into existence rapidly, although not with uniform intensity, over a large extent of the area of a fibre bundle seems only in accordance

with what might be expected. We see then that in the initial stages of glaucoma the field may be normal or show slight depression, with or without an early nasal step or arcuate defect. These changes are variable and inconstant and may not be elicited on every occasion.

In the next stage these transitory conditions become permanent. While the tension and with it the field changes vary from time to time the recessions, as regards the field, do not reach the normal level. The initial depression persists but does not increase much. A depression which is more pronounced peripherally sets in, more especially at the periphery of the affected quadrants, so that while central vision remains unaltered, the nasal field shows signs of commencing restriction. This process advances at first more slowly than the loss of field due to the growth of the arcuate scotoma.

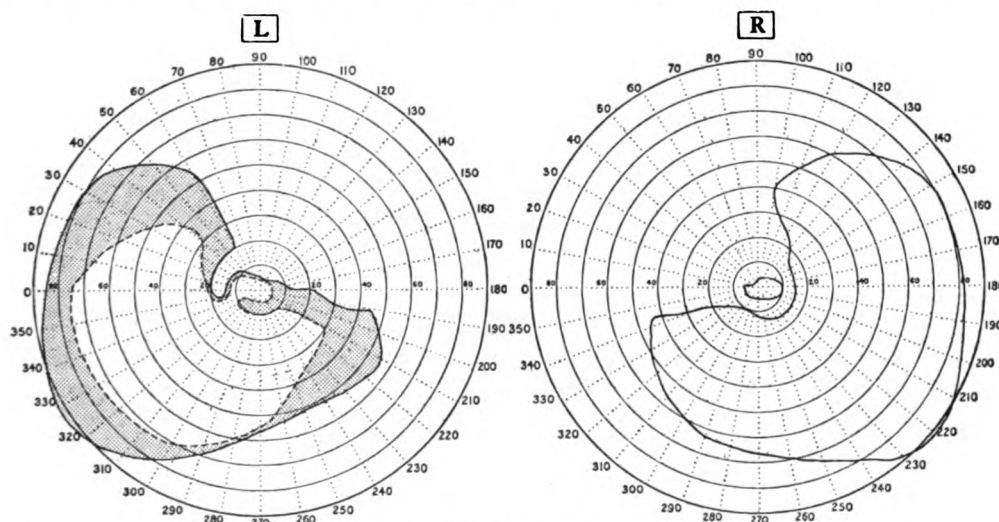


FIG. 71.—GLAUCOMA.

Showing isolation of central field, nasal step and peripheral nasal loss. The isolation is incomplete in the left field for the objects used, $33\frac{5}{8}$, $33\frac{3}{8}$. R.V. $1\frac{6}{8}$. L.V. $6\frac{6}{8}$. Note small central nasal step in R. central field. (S., 1921.)

This scotoma now develops its characteristic appearance, usually in the upper half of the field, arching over the fixation area to the nasal step. As it increases it gradually widens on both sides, more especially on that away from the fixation area, so that the nasal step and the restriction of the nasal field in the affected quadrant become more pronounced (Fig. 70). Soon it becomes doubled, foreshadowing the nerve fibre bundle ring scotoma with isolation of the centrocæcal area. In the more affected quadrant, more often the upper, the arcuate scotoma widens into a sector defect and the field is rolled or rotated, as it were, temporalwards producing a somewhat irregular horizontal hemianopia. When the failure is more equal in the upper and lower halves of the field the centrocæcal area, including the fixation area, becomes isolated from the rest of the field and remains surrounded by a ring scotoma, which usually opens out or breaks through on the nasal side. It is this breaking through of the nasal side of the arch of the nerve fibre bundle ring scotoma, together with the loss of the areas of the peripheral fibres of the arching bundles in the upper and lower nasal quadrants of the field, which

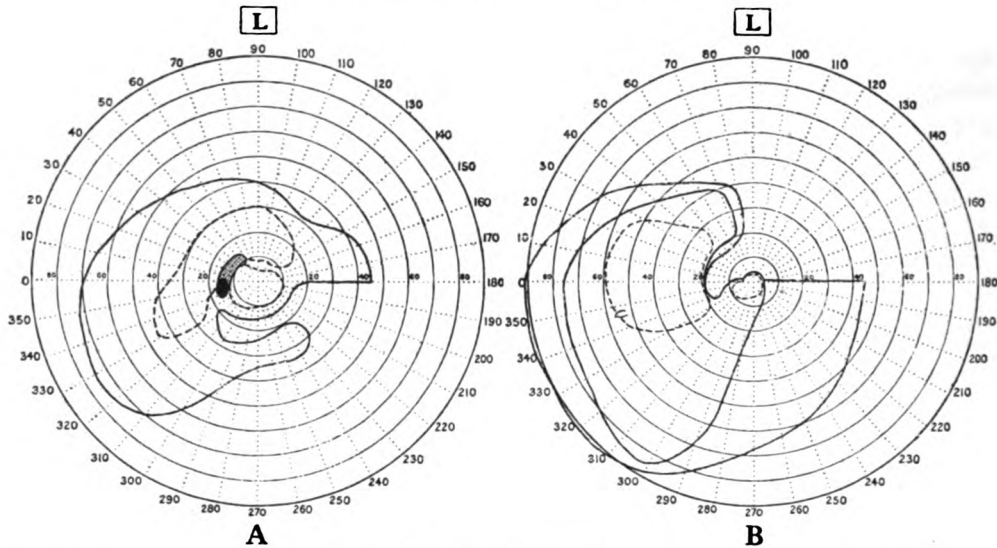
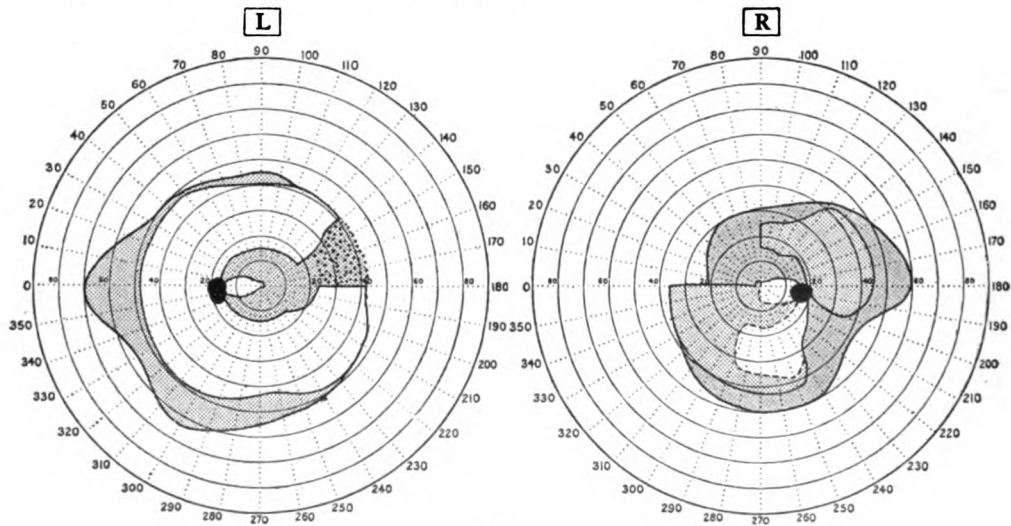


FIG. 72.—GLAUCOMA.

- A. Showing isolation of central field and nasal step. The arcuate scotoma above has its denser part adjoining the blind spot, the lower one is denser peripherally. Objects $3\frac{3}{30}$, $3\frac{3}{30}$. V. $3\frac{3}{30}$. (D., 1919.)
 B. Illustrates the retention of the central and temporocæcal areas. The nasal step is present for a large object ($3\frac{3}{30}$) only. Objects $3\frac{3}{30}$, $3\frac{3}{30}$, $3\frac{3}{30}$. V. $1\frac{6}{12}$. (O.C., 1922.)



GLAUCOMA.

FIG. 73.—SYMMETRICAL ARCULATE DEFECTS AND NASAL STEPS.

The test-object ($3\frac{3}{30}$) was dimly seen in the dotted area. Note cuneate defect in the right temporal field. R. V. $2\frac{4}{24}$. L.V. $3\frac{3}{30}$. Objects $3\frac{3}{30}$, $1\frac{6}{12}$, $1\frac{6}{12}$. (H., 1922.)

has given rise to the traditional "failure of the nasal field" associated with glaucoma. This nasal "contraction" is not so much due to a centripetal shrinking of the nasal peripheral field as to the continued centrifugal expansion of the nerve fibre bundle defects. By this time the peripheral depression is becoming evident in the temporal field by the contraction of the isopters, which begin to shrink, although the more central part of the temporal field, near and especially on the temporal side of the blind spot,

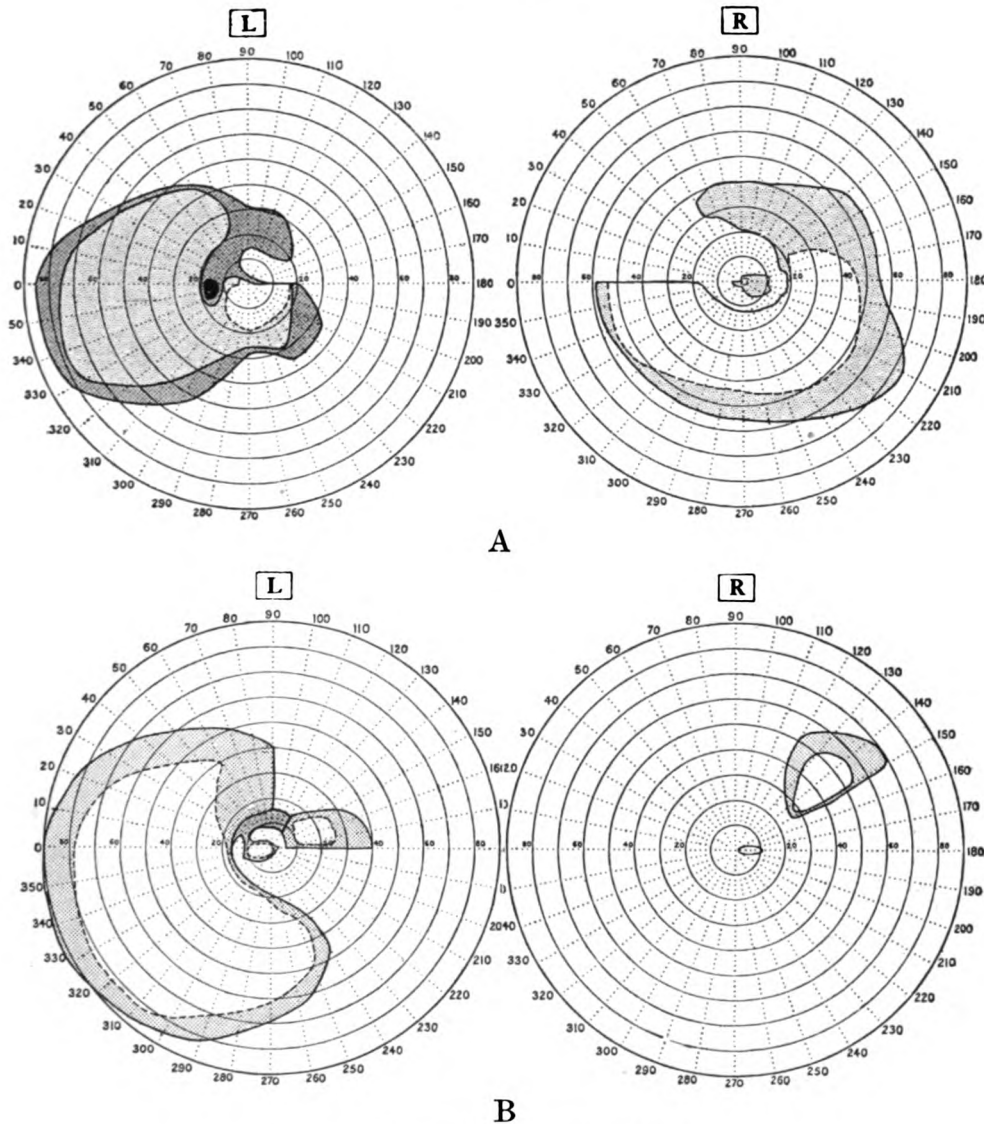


FIG. 74.—TYPICAL GLAUCOMA FIELDS

- A. R.V. $\frac{6}{6}$. L.V. $\frac{3}{6}$. Small central nasal step in R. field. In the left field the dense part of the arcuate scotoma does not extend to the blind spot. (A., 1923.)
- B. R.V. $\frac{6}{6}$. L.V. $\frac{3}{6}$. Irregular loss of upper nasal quadrant in left field: preservation of nasal patch. Later this became isolated. Note the patch of retained vision upwards and temporally from the blind spot in the characteristic position in the right field. (E., 1924.)

remains relatively undepressed. Temporal nerve fibre bundle defects are not common, and a straight Bjerrum scotoma, in the form of an isolated, narrow, sharply defined, cuneate defect extending from the blind spot horizontally to the temporal periphery, is unknown, although in those cases in which the field assumes the form of a horizontal hemianopia it is often evident that the straight margin of the field corresponds to the radial direction of the nasal fibres. The peripheral loss now proceeds more rapidly than the central, so that while the oval centrocæcal area shrinks slowly the remaining parts of

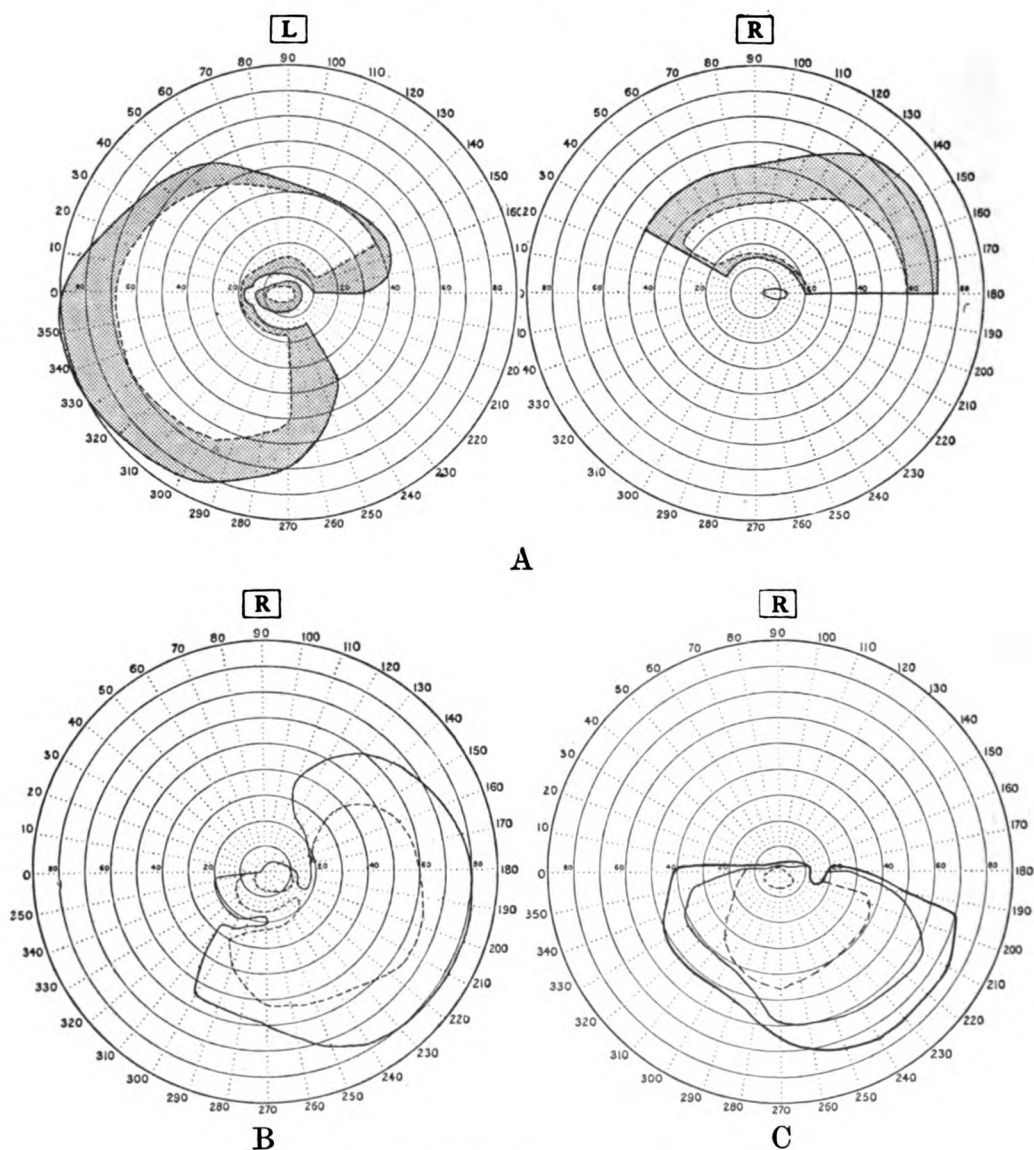


FIG. 75.—TYPICAL GLAUCOMA FIELDS.

- A. Right field shows preservation of upper outer quadrant and a minute field in centrocæcal area. V. $\frac{5}{6}$ slowly. Left field shows superior and inferior arcuate defects forming a ring scotoma and breaking through to nasal periphery. V. $\frac{5}{6}$. (S., 1923.)
- B. Double arcuate defects with nasal step and loss of nasal field. The inferior arcuate defect does not extend to blind spot. V. $\frac{5}{6}$. (A., 1925.)
- C. V. $\frac{5}{6}$ +. Right field of same case as Fig. 59 A. Horizontal hemianopia. (M., 1924.)

the field gradually disappear, until all that survives is a patch to the temporal side of and near the blind spot, while central vision is still relatively well preserved. The diminished centrocæcal area now becomes cut off from the blind spot, and then the fixation area is lost, leaving a small narrow isolated horizontal strip between the fixation point and the blind spot (Figs. 74 B, 75 A). This disappears, and then lastly the area outside the blind spot. Central vision persists until a late stage, and it is noteworthy that, although

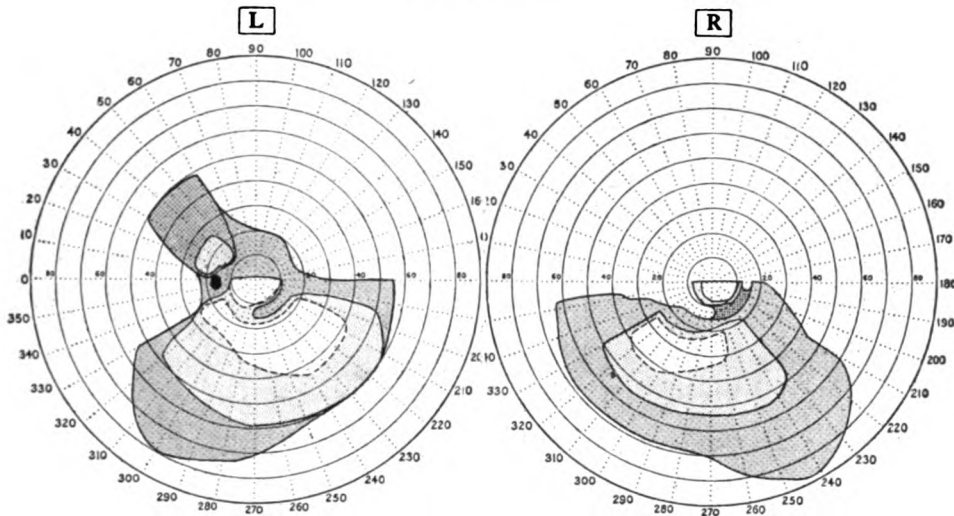


FIG. 76.—ADVANCED GLAUCOMA. HORIZONTAL HEMIANOPIA.

R.V. $\frac{6}{6}$. L.V. $\frac{6}{6}$. In both fields the denser part of the inferior arcuate defect stops short of the blind spot. (A., 1924.)

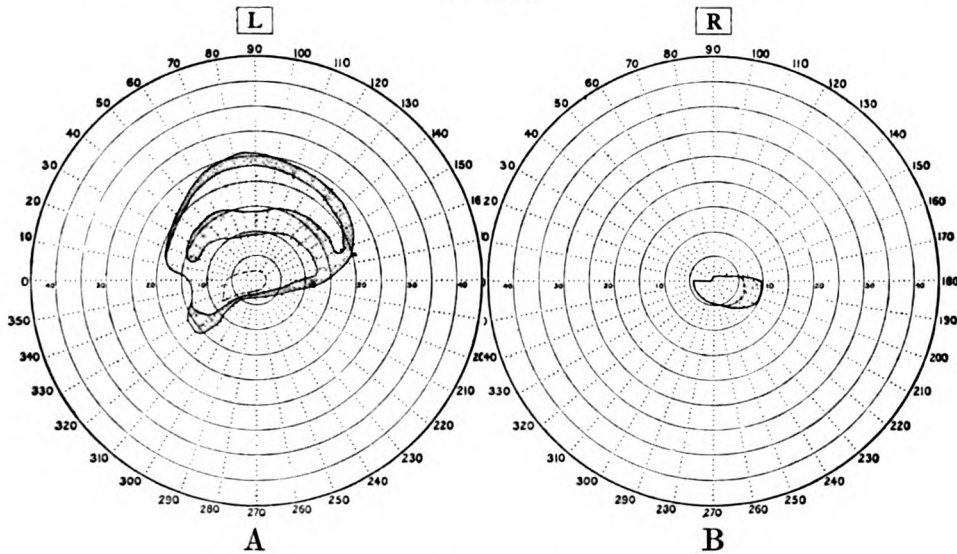


FIG. 77.—ADVANCED GLAUCOMA. TERMINAL STAGES.

Charts on larger scale. V. $\frac{6}{6}$ in each case. In B the periphery has entirely disappeared before the central area. Note minute central nasal step.

Objects A. $\frac{2000}{2000}$, $\frac{2000}{2000}$, $\frac{2000}{2000}$. (T., 1924.)

B. $\frac{2000}{2000}$, $\frac{2000}{2000}$. (H., 1924.)

the centrocæcal area often persists in an almost stationary condition for a considerable period while the peripheral loss is increasing, when the end comes it is practically always survived by a more peripheral temporal patch.

These late changes can only be studied in eyes which are nearly blind.

The way in which the fixation area becomes invaded and obliterated is of interest, although it has not yet been widely studied. Since the fovea lies at the central end of the horizontal raphé of the retina, and is mainly supplied by very slightly curved fibres

C.F.

K

arching towards it from above and below, central vision cannot be obliterated by damage to the fibres at either the upper or the lower side of the disc alone, which would at most result in a horizontal division of the nasal part of the fixation area. The termination of the arcuate scotoma lies in the first instance always on the horizontal meridian to the nasal side of the fixation area. As the scotoma widens it advances extremely slowly towards the fixation point and relatively much more rapidly towards the periphery. The development of this process can often be traced in the formation of a minute central nasal step. Ultimately the defect has a straight edge from the nasal periphery to the fixation area, corresponding to the retinal raphé, while from the fixation area to the blind spot the edge has a slight concave curve corresponding to the direction of the fibres. The fixation area is "spared," and only exceptionally does it become transversely divided by further failure

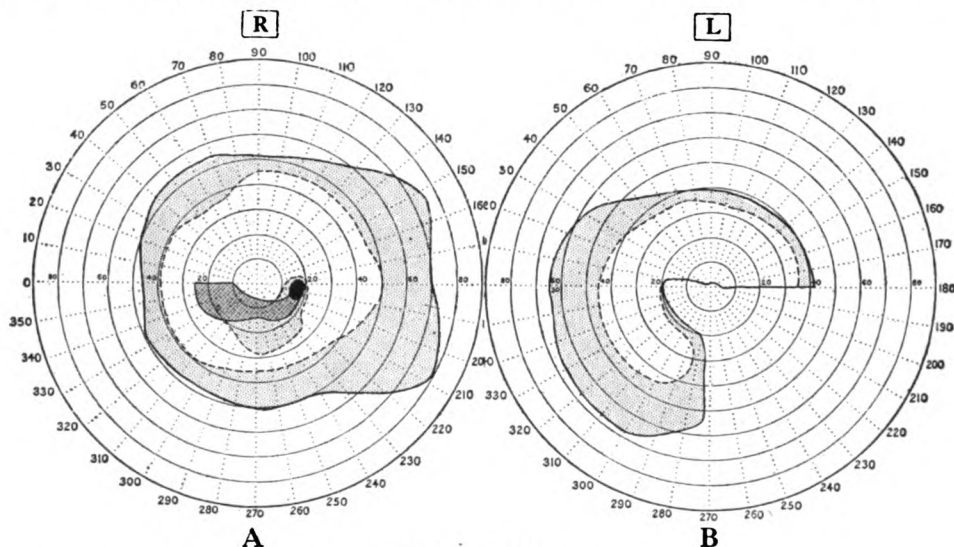


FIG. 78.—GLAUCOMA.

- A. Arcuate scotoma with little peripheral change. V. $\frac{6}{6}$. Objects $\frac{5}{30}, \frac{1}{30}$. (K., 1913.)
 B. Early central loss. V. Fingers at 1 m. Objects $\frac{5}{30}, \frac{1}{30}$. (Kr., 1913.)

in the same direction. Almost always, owing to a similar process on the opposite side of the horizontal meridian of the field, the centrocaecal area becomes isolated, forming a horizontally oval island of vision having the fixation area just within its nasal end and the blind spot in contact with its temporal end. The nasal end frequently shows a small nasal step. This island becomes narrower, becomes cut off from the blind spot, and then shrinks away from the fixation area, so that central vision is lost. The last remnant of the central field then lies between the blind spot and the fixation point, nearer the latter (Figs. 74 B, 75 A). Occasionally, before the loss of the fixation area, a small isolated scotoma appears upon its temporal side. Finally, the minute narrow strip remaining disappears.

Occasionally the central part of the field fails early, leaving the periphery intact for a considerable time and thus gives rise to an oval centrocaecal scotoma which surrounds the fixation area. In such cases the defect has developed from two small symmetrically opposed arcuate scotomata originally meeting on the horizontal meridian to the nasal side

of the fixation area, forming a ring scotoma around the centrocaecal area, which becomes isolated in the usual way. Then, without increasing much on its convex aspect, the scotoma enlarges towards the horizontal meridian, and the island of vision is lost as already described. In some of these cases a peculiar cylindrical, rather than goblet-shaped, cupping with relatively intact disc periphery may be noted. Central vision is apparently never lost by the early formation of a nasocæcal scotoma encroaching upon the fixation area from the temporal side.

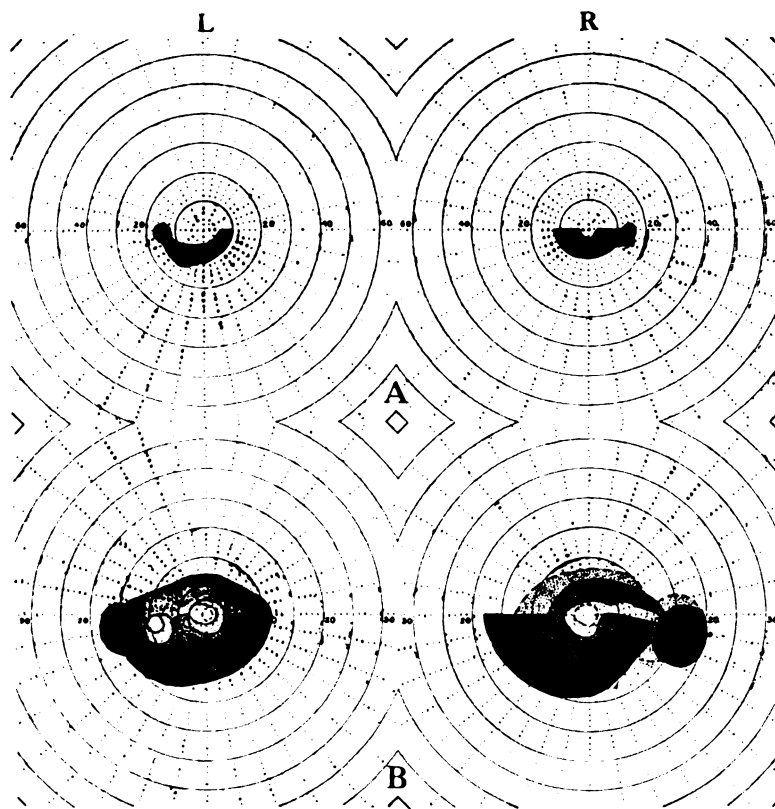


FIG. 79.—GLAUCOMA. CENTRAL DEFECTS WITHOUT PERIPHERAL CHANGE, ILLUSTRATING THE VALUE OF SCREEN ANALYSIS.

The peripheral fields were entirely normal, and the perimeter findings (A) give very imperfect information as to the true state of central and paracentral vision. Some resemblance to the centrocaecal scotoma of retrobulbar neuritis is present, but analysis shows that each scotoma is composed of opposed arcuate defects. R.V. $\frac{1}{2}$. T. 35. L.V. $\frac{1}{2}$. T. 47. Disc cupped centrally.

Objects:—(A) R. $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ L. $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$.

(B) $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$.

Central colour vision normal. (F., 1923.)

All these changes, both peripheral and central, develop with a strong tendency to symmetry in the two fields, and often also in the upper and lower halves of the same field. At the same time no definite or rigid sequence is followed and the defects are always in different stages, so that while the description given corresponds to a common and usual form of development, numerous variations occur, and in a series of cases a great number of different appearances and phases of the nerve fibre bundle defect, all of the same basal type, will be found.

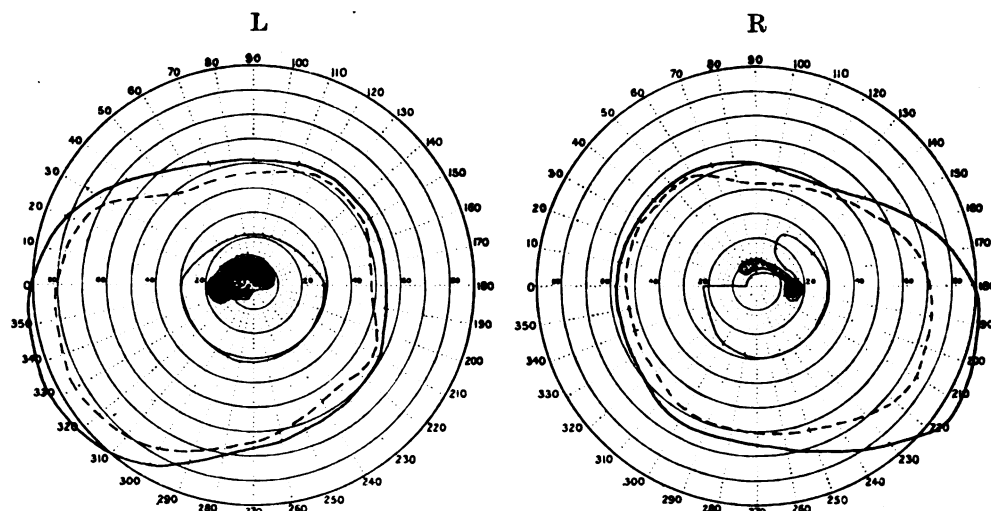


FIG. 80.—GLAUCOMA.

Right eye: shows typical conditions in centre of field; arcuate scotoma for 2000 and 2000 . Periphery normal. Objects 2000 , 2000 , 2000 ; 330 , 330 . V. $\frac{1}{2}$. T. 25. Disc widely cupped.
 Left eye: intense central defect with minute centrocaecal field for 2000 . Periphery normal. V. $\frac{1}{2}$. T. 18 (previously 45). Disc cupped in lower outer third only. Objects 2000 , 2000 ; 330 , 330 . (McC., 1930.)

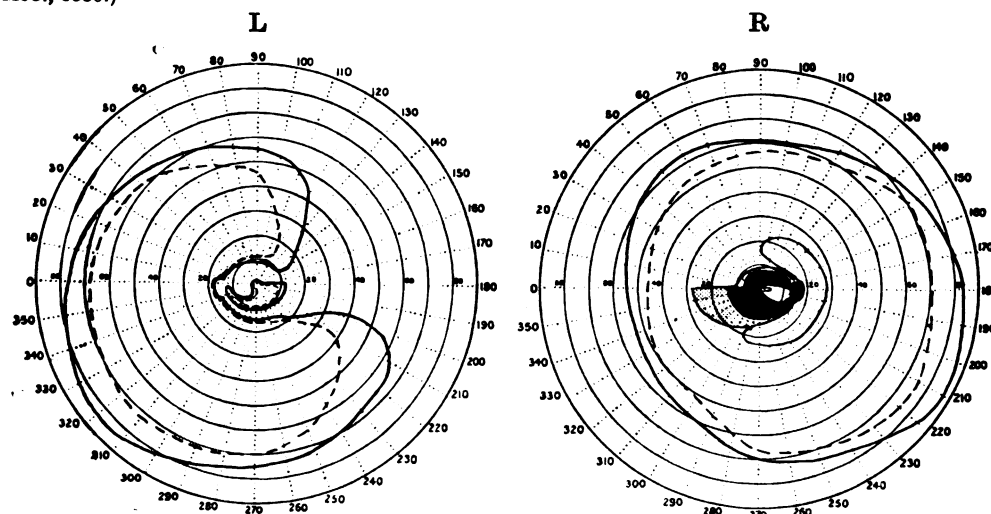


FIG. 81.—GLAUCOMA.

Right eye. Central defect without peripheral change. V. $\frac{1}{2}$. T. 18. Disc deeply cupped. Has undergone operation. Objects 2000 , 2000 , 2000 ; 330 , 330 .
 Note very small central field for 2000 , nasal step for 2000 and 2000 .
 Left eye. Here the central defect has broken through to the periphery on the nasal side, but the centrocaecal area has given way in an atypical manner.
 V. $\frac{1}{2}$. T. 18. Disc deeply cupped. Has undergone operation. Objects 2000 , 2000 ; 330 , 330 (D. V., 1930.)

The uses of perimetry in glaucoma may be summarised as follows:—

1. In throwing light upon the mode of production of the visual defects.
2. In diagnosis.
3. In prognosis.
4. In helping to indicate the form of treatment, and in observing its effects.

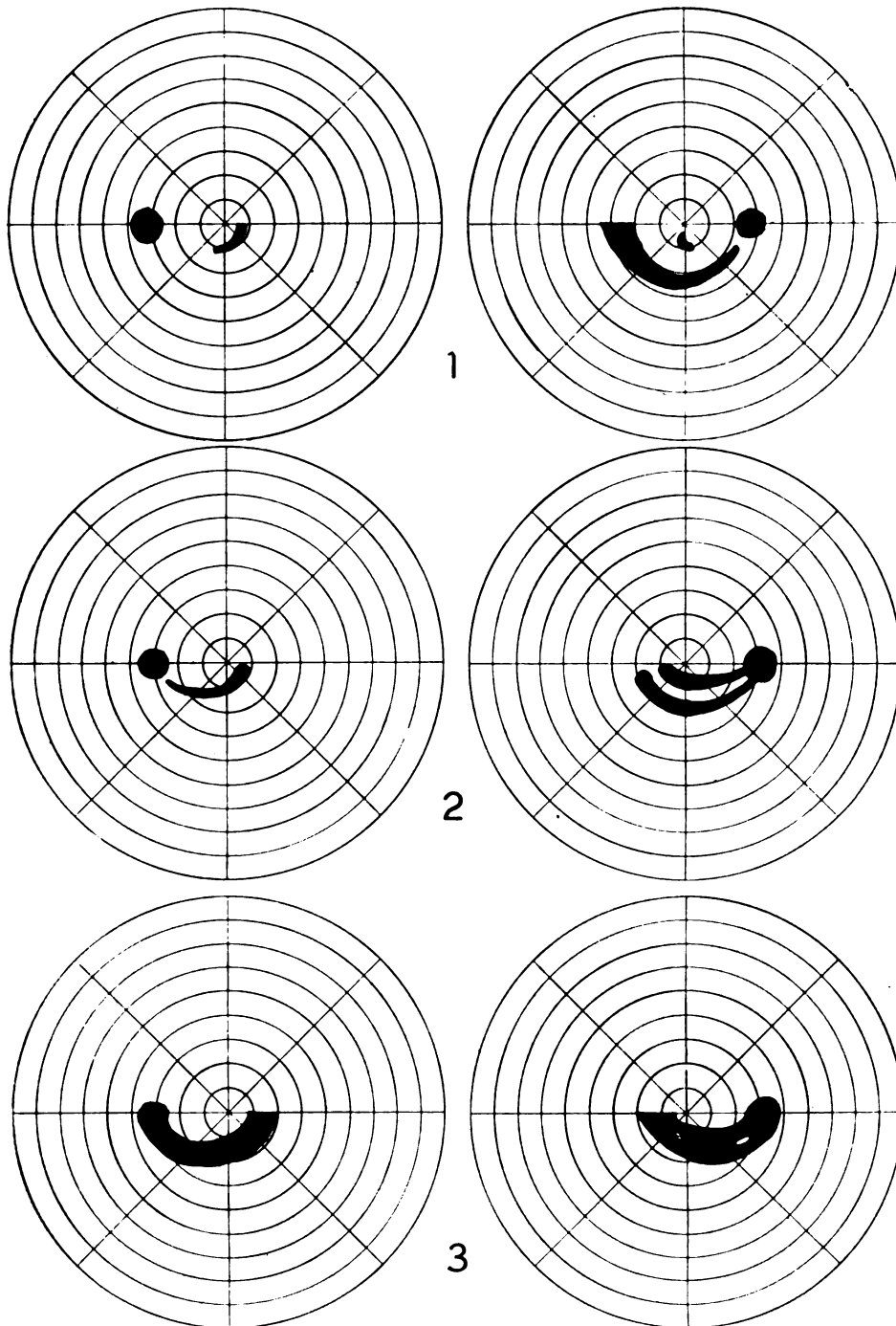


FIG. 82.—DEVELOPMENT OF NERVE FIBRE BUNDLE DEFECT TOWARDS BLIND SPOT. (DALSGAARD-NIELSON, 75).

Courtesy of the editor and publisher of the *Trans. Ophth. Soc. U. K.*

Interpretation.—It must be admitted that the cause of the general depression of the field which is present at an early stage is obscure. It does not increase to the degree that might be expected if it were a progressive process in itself apart from the development of the nerve fibre bundle defects. This is shown by the fact that in the late stages vision for colour as well as for white is usually only slightly impaired in the better parts of the retained areas. It would appear to be an expression of lowered functional efficiency depending on the tension, and it is unnecessary to discuss here the various theories of retinal compression, malnutrition, etc., none of which is completely satisfactory. The effect of a very small pupil should not be forgotten.

It has been suggested that the contraction of the nasal side of the field might be explained by the assumption that the nerve fibres and retinal vessels passing temporalwards are, on account of their greater length, more liable to damage from increased intraocular pressure than those which pass nasalwards. Apart from other reasons the evidence of perimetry strongly indicates that it is the anatomical conditions of the disc, and not the length of the fibres between the disc edge and the retinal periphery, which determine the order in which the fibre groups suffer. Perimetric studies show that the first fibres affected are nearly always those which enter the disc to the lateral side of its upper and lower poles, more often the lower, successive fibres to the nasal side of these groups are then involved and the papillo-macular bundle usually very late. It appears to be the fibres in the neighbourhood of the large vascular trunks which are most susceptible. The tendency to symmetry in the fields is evidently a reflection of anatomical symmetry in the discs. Undoubtedly the formation of the glaucoma cup is an important factor, but this is the result of pressure acting in each case upon a certain form of disc so that different varieties of cupping depend upon details in the structure of the disc present before the pressure began to act, while the degree of pressure and the duration of its action probably influence the amount, rather than the kind, of cupping.

The study of the arcuate scotoma does little to solve the problem of the structure of a nerve fibre bundle. Assuming that the most peripheral fibres lie most superficially we may suppose that where an arching bundle traverses the vertical meridian of the retina the crossed fibres apply themselves to the deep surface of the uncrossed, and then both pass the margin of the disc together, diverging later to their appropriate positions in the nerve. If we also assume that the fibres next to the edge of the scleral foramen are most liable to damage, we should expect to find that the arcuate defect begins at the edge of the blind spot and gradually extends peripherally. The mode of development of the field changes affords little support for these assumptions. Although the defects begin near the blind spot they do not creep out from it, but towards it, nor is the most intense part of an arcuate scotoma always that which is nearest the blind spot (Figs. 75 B, 76), and after successful operation a scotoma, previously continuous with the blind spot, may recede from it, showing that the retinal area nearest the blind spot has recovered more than that more remote. In the temporal field the failure is mainly peripheral from the first, and temporal extensions from the blind spot are uncommon, while the final remnant of the field is often close to the temporal side of the blind spot with which it may remain in

contact until a very late stage. For these reasons it is clear that the frequently advanced explanation of the production of field defects in glaucoma by compression of the nerve fibres against the edge of the scleral foramen cannot be regarded as satisfactory.

The evidence of the scotomata suggests that it is not the nerve fibres but their blood-vessels which are compressed, in the first instance at any rate, and that in this way the precise nature of the defect is determined. The collateral circulation of the circum-papillary retina has been referred to on p. 298. On this hypothesis the appearance of an arcuate scotoma at some distance from the blind spot may be explained by assuming that one of the arterial twigs passing to the group of fibres concerned was so related to the disc edge as to become stretched or kinked and so more or less occluded.

The absence of disproportion between the defect for colour and that for white indicates that as the nerve fibres become involved they are severely affected. There is little evidence of the partial impairment of a considerable percentage of the fibres in an affected group in the early stages, but rather of the severe impairment of a small proportion or of a narrow bundle. In the central field the lesion appears to involve the fibres in small groups, suppressing the function of each before the next is included. This is well shown in chronic cases by the steepness of the edge of the arcuate defect in the central part of the field. There may be quite a narrow band of absolute blindness only 2° or 3° in width passing to the blind spot, bounded on each side by areas having almost normal visual acuity. Such a band represents a fibre group about twice as wide as a retinal vein at the disc, completely blocked, while the fibres immediately adjoining are practically unharmed. The explanation is probably anatomical and concerned with the vascular supply of the nerve fibres.

The correlation of the disc changes with the field defects presents an interesting and difficult problem. Some observers have found that the area of most pronounced field change corresponds to the part of the disc where the cupping appears greatest. In most cases, however, neither the position nor the degree of the field changes can be inferred from the cupping, though, on the whole, extreme cupping and especially extreme pallor are associated with severe field defects.

In eyes which are definitely glaucomatous, that is to say, in which raised tension and other suggestive indications are present, the disc may be apparently normal, or may show quite definite early pathological cupping without any field change beyond baring of the blind spot or slight depression, or sometimes even without any demonstrable field change whatever. Unless exhaustively tested by the quantitative method, these slightly depressed fields are indistinguishable from normal fields, and undoubtedly in the past they have frequently been recorded as normal. The appearance of any form of arcuate defect may be regarded as evidence, possibly the first evidence, of definite localised conduction interference at the disc edge and is, in my experience, nearly always associated with pathological cupping or pallor. If cupping cannot be discerned and an arcuate defect is present, other possible conditions must be carefully considered, and the evidence from other sources must be strong before the defect can be interpreted as the result of glaucoma. If the case is really one of glaucoma, the development of visible cupping cannot be long delayed when once nerve fibre bundle defects have begun to

appear. Unfortunately this question is complicated by the personal equation of the observer in regard to ophthalmoscopic appearances.

In the absence of cupping typical, though never well-developed, field changes may be present, but only in very rare cases, while pathological cupping is almost always associated with field changes.

Pallor of the disc is important in regard to the presence of field changes, in the lesser degrees perhaps even more important than cupping. The relationship may be expressed somewhat as follows :—

If there is neither cupping nor pallor, field changes are absent or doubtful.

If there is cupping without pallor, field changes may be absent or very slight.

If there is pallor without cupping, characteristic field changes are present.

If there is cupping with pallor, well-developed field changes will be found.

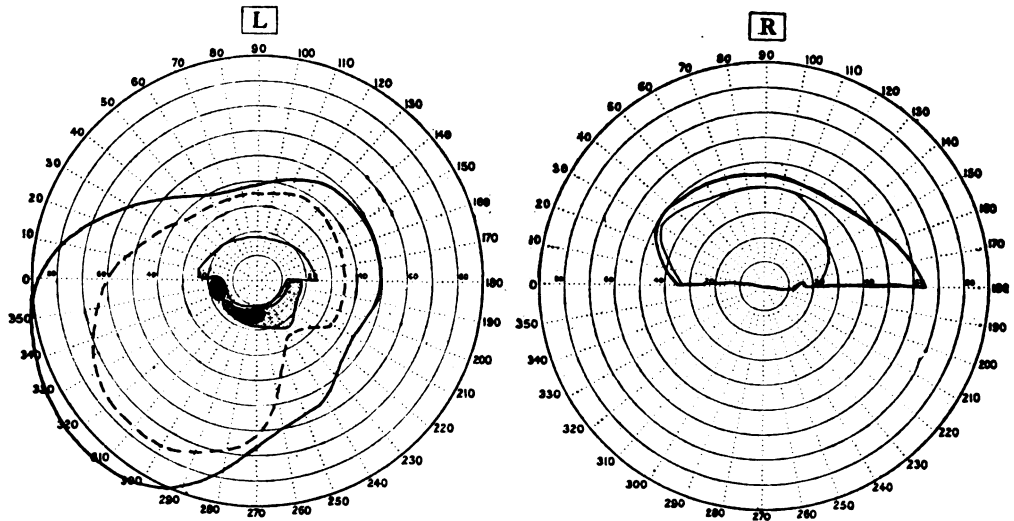


FIG. 83.—GLAUCOMA WITHOUT CUPPING.

Both optic discs were slightly pale; the right showed a minimum degree of cupping, the left none. R.V. $\frac{5}{8}$. L.V. $\frac{8}{8}$.

Objects R. $\frac{3}{30}$, $\frac{3}{30}$. L. $\frac{3}{30}$, $\frac{3}{30}$, $\frac{2}{30}$. (A., 1926.)

The relation of the field defects to the tension would appear to be mainly governed by the effect of the intraocular pressure upon the nerve head. Little is known with regard to the length of time during which increased intraocular pressure must act before either cupping or field changes are produced. Probably it extends over a very variable period of months or years according to the degree of pressure and the resistance of the disc structure. In one case tension of 70 Schiötz existed for some months producing wide cupping but no pallor and only slight central depression of the fields. In another case symptoms existed for ten years, causing cupping but not pallor and only very early field changes. On the other hand in glaucoma following incarceration of the iris cupping and pallor with severe field changes may develop rapidly. Nor is there much evidence as to the precise character of the field changes, if any, produced in the initial stages by a rise of tension of short duration. The question is complicated by the presence of corneal oedema when the tension is considerable. With a slight rise

there may be no field change, or merely a low degree of depression which disappears when the tension becomes normal again. Arcuate defects which come and go with the rise and fall of the tension have been reported, but I have not yet been able personally to confirm this observation. It is, however, certain that lowering of the intraocular pressure by meiotics or operation can cause a very considerable recession of the field defects, especially in the early stages. It is very often in connection with relatively low tension of long duration that the most advanced field changes are found; symptoms other than visual are absent or few, and the patient does not seek advice until a late stage.

These and other problems aroused by the perimetric study of glaucoma present wide scope for investigation, and, in the meantime, may be regarded with an open mind.

Diagnosis.—Since the field changes are the result of the glaucomatous process, and by no means necessarily an early result, it is evident that their detection, even in their earliest form, does not indicate that the disease is commencing, but that it is already established. The recognition of glaucoma in what may be called its pre-perimetric stage is developing, and, as our knowledge and skill in this respect increase, we may expect to find that the elicitation of the field changes is mainly useful in supporting and confirming, rather than in initiating, the diagnosis. Moreover, most cases apply for advice when the other signs are so distinct that the question raised is not whether glaucoma is present, but how much damage has already been done. This question cannot be adequately answered without the aid of perimetry. Apart from confirming an opinion already arrived at, perimetry has a definite diagnostic value in certain cases in which the other signs are doubtful or insufficient. We may want to know whether the apparently sound eye, where one is glaucomatous, is affected, or whether a suspicious-looking disc, not associated with increased tension, is accompanied by field changes.

The less prominent the other features of the clinical picture the more important is perimetry in diagnosis and the greater the necessity for careful perimetric examination; nevertheless a positive diagnosis based on field changes alone should be made only with the greatest caution.

From the point of view of perimetry these cases of possible or suspected glaucoma may be considered in two chief groups according as gross field changes are present or not.

GROUP I. No obvious gross field changes or definite loss of vision are present.

- A. Neither symptoms nor signs are present. The suspected eye appears in every way normal and healthy, but is suspected because definite glaucoma is present in the other eye.
- B. Symptoms alone are present, *e.g.*, periods of ocular discomfort, halos, or other suggestive indications but both disc and tension appear normal.
- C. Ophthalmoscopic signs alone are present. Either cupping or pallor of the disc, alone or together, found during routine examination, is the only suspicious sign.

D. Increased intraocular pressure alone is present. The tension is found to be raised but no other symptom or sign is elicited.

GROUP II. Gross and easily detected field changes are present.

Cases in this group all present well-developed features especially in the optic disc. Characteristic changes in the optic disc and raised tension are present in every case and the problem is not the detection of early signs but the proper interpretation of the well-developed changes.

In the first group careful search must be made for the very earliest signs such as depression of the central field or baring of the blind spot. These signs may or may not be elicited. Cases falling under sub-group C are the most likely to give positive results. Only the central part of the field need be examined and the stimulus must be reduced as far as possible by diminishing the size of the object, increasing its distance from the eye, and lowering the illumination by darkening the room. All three methods may be used together.

The screen at 2 m. is used and the blind spot is first mapped out with $\frac{40}{2000}$. The isopter for $\frac{1}{2000}$ is then examined using both circular and radial movements and paying special attention to its relation to the blind spot and to the horizontal meridian on the nasal side. The amblyopic zone of the blind spot should be carefully demarcated. The isopter for $\frac{2}{2000}$ is then marked out to see how far it shares in any changes found for $\frac{1}{2000}$. Somewhat larger test-objects may be used if necessary but are not often required in cases belonging to Group I.

Finally, isolated scotomata are sought for, more especially about the line of the 15° circle between the blind spot and the vertical meridian. If no defect is found, the examiner has been unable to elicit perimetric evidence of glaucoma on that day, and in the absence of other signs the eye may be regarded as normal.

In examining the poles of the blind spot care should be taken not to be misled by the normal defects present here, due to the large vessels. These defects are only elicited by careful search with small angles such as $\frac{1}{1000}$ or $\frac{2}{2000}$, they are wider near the blind spot and become narrower as they leave it, their curvature does not correspond to that of the nerve fibres, they are present at both poles of the blind spot and they are somewhat indefinite; the test-object "goes in and out," probably owing to slight unsteadiness of fixation. Their relation to the vessels may be checked by the ophthalmoscope. A short distance from the blind spot this kind of defect splits up into branches representing the large vascular trunks. It does not really simulate a true arcuate scotoma.

Depression of the upper part of the central field with baring of the blind spot and a small central nasal step may be regarded as conclusive evidence of glaucoma in Group I. Baring of the blind spot alone is very suggestive but not conclusive; depression with a nasal step alone is a more definitely positive sign.

The presence of arcuate defects indicates that the disease has advanced to the stage of damage to the fibres; their absence in no way contra-indicates the diagnosis of glaucoma.

It has already been pointed out that the *shape* of nerve fibre bundle defects depends upon anatomical, and not pathological factors. The characters of such defects are, nevertheless, modified by the nature of the cause. While it is true that arcuate defects, if shape and position only are considered, are, as was originally pointed out by Bjerrum, in no way peculiar to glaucoma, similar field changes occurring in other diseases do not analyse in the same way and can hardly be confused with those of glaucoma if observation is adequate. Some of these defects have already been mentioned, and others will be referred to later. Nor should it be assumed that a scotoma which arches from the blind spot is necessarily a fibre bundle defect or Bjerrum scotoma on this ground alone.

Knapp (221, 222) has pointed out that optic atrophy with excavation of the disc resembling glaucoma may be due to sclerosis of the cerebral basal vessels but in these cases the field changes are not of the glaucoma type.

If, for any reason, glaucoma is suspected in an eye when it is not present in the opposite eye, and when the disc is pale but does not appear to be pathologically cupped, and no increased tension can be detected, the presence of field changes of the Bjerrum type can be accepted as evidence of this disease only after the most careful exclusion of other possible causes of nerve fibre bundle defects and the consideration of the case as a whole.

It may, however, be safely said that an arcuate scotoma, as described in the preceding pages, which conforms in *all* its characters to the glaucoma type, is never produced by any other disease.

The great majority of cases belong to Group II. Here the signs of glaucoma, apart from field changes, are well developed by the time that the patient seeks advice, and the examination of the field is then directed towards ascertaining the amount of damage which has been done and the outlook for the future. In this group the periphery of the field should be investigated with objects of 3 mm. to 5 mm. or more at 330 mm., and then with $\frac{1}{330}$. The central field is then examined and a composite chart made up. Perimetry is of assistance in identifying the field changes with the glaucoma type, in estimating the damage already done, in suggesting the form of treatment preferable, and in prognosis rather than in diagnosis.

Prognosis.—It is in regard to prognosis that perimetric studies are especially valuable. With few exceptions, the worse the field the worse the eye, and vice versa; the results of operation are much less satisfactory, on an average, when the field is already in a bad state.

The bearing of the condition of the field upon treatment is of interest to the clinician. Apart from other factors such as the condition of the eye and the age and health of the patient, the extent and intensity of the field changes and the proximity of the defect to the fixation area are of great importance. Thus a very restricted field in an old person may indicate treatment by meiotics in preference to operation. If the edge of the defect is found to be sloping at any part, or if areas of doubtful vision are present, a progressive condition should be suspected, as the sloping edge may be found to recede and the weak or doubtful area to become definitely scotomatous. On the other hand, a steep or precipitous margin indicates that the defect is relatively stationary or only slowly progressing

at that part. The behaviour of the defect near the fixation area should be carefully watched, as the possibility of loss of central vision is a constant source of anxiety to the surgeon. But, as we have seen, in the later stages of slowly advancing chronic glaucoma the peripheral loss progresses more rapidly than the central, and the centrocæcal area is often surrounded by a steep-edged arcuate or ring scotoma, and usually remains relatively well preserved. It is, therefore, in the peripheral field, unless the changes are limited to the central area, that the progress of the disease is most easily observed, and it is here that a watch should be kept for indications as to procedure. For this reason it must be emphasised that in the study of glaucoma, and also in other conditions, the screen is complementary to, but does not supplant, the perimeter and should not be used alone.

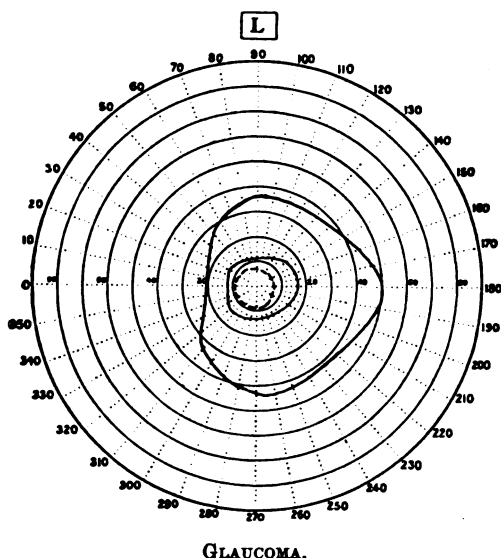


FIG. 84.—CONCENTRIC CONTRACTION TWO AND A HALF YEARS AFTER OPERATION.

Small concentrically contracted field, steep-edged, without characteristic features of glaucoma. Objects $\frac{3}{30}$, $\frac{4}{30}$, $\frac{5}{30}$. V. $\frac{3}{8}$ T. 5. Disc. atrophic, but not cupped. (F., 1936.)

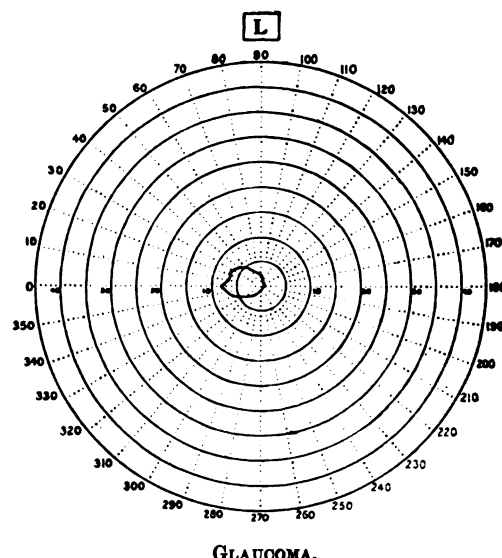


FIG. 85.—CONTRACTION FOLLOWING OPERATION.

Field of left eye for $\frac{3}{30}$, reduced to less than 10° . No larger field obtained with larger objects. V. $\frac{1}{18}$. T. 11. (B., 1927.)

It is sometimes maintained that if the tension be watched the progress of the case and the need for surgical intervention may be correctly estimated without recourse to perimetry, or at least to detailed investigation of the field; in other words, that, if the tension is kept down, the field will look after itself. This attitude should not be adopted without reserve. It is precisely in those cases in which the tension is relatively low that perimetry is of the greatest value, and it is impossible without its aid to be sure that the defects are not increasing.

When the scotoma is beginning to invade the fixation area, operation, especially if it includes iridectomy, has been regarded as dangerous by some surgeons. There is no evidence that it is the proximity of the defect to the fixation area which constitutes the danger. On the other hand, as Brandt has shown, the cases with most advanced field defects are less likely to do well after operation, and it is in such cases, as a rule, that the

fixation area is most closely threatened. Other surgeons believe that the more closely the fixation area is approached the greater the need for immediate operation. Van der Hoeve's reported cases show that, whether the peripheral field be good or bad, a scotoma closely approaching the fixation point does not contra-indicate trephining, which he believes to be preferable to iridectomy or operations involving iridectomy, when central vision is threatened. It seems reasonable to suppose that when the fixation area is attacked from one side only, it is less endangered than when menaced from several directions, as horizontal bisection of the fixation area is a rare event. It is well to remember in this connection that a threatened central area may live long, it may be for years, and in any case the course adopted will depend not only on the state of the field, but upon

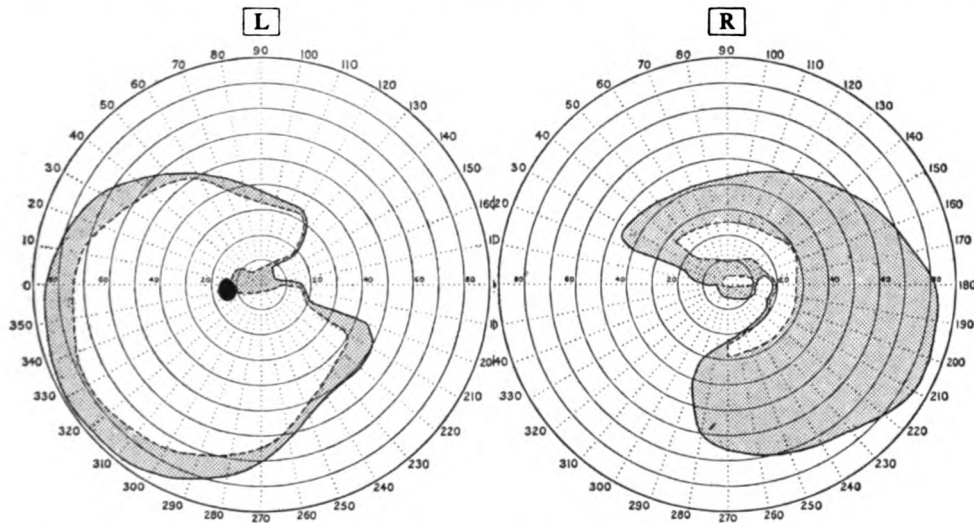


FIG. 86.—GLAUCOMA WITH CENTRAL CHOROIDO-RETINITIS.

In the right eye the glaucoma was advanced and the retinal lesion slight, causing loss of the lower part of the central field. In the left eye the glaucoma was in a relatively early stage, but the retinal lesion severe enough to cause great depression of central vision. R.V. = $\frac{8}{8}$ —. L.V. = $\frac{8}{80}$. (L., 1924.)

the whole clinical picture and the discretion of the surgeon. My personal experience is entirely in favour of operation when the fixation area is threatened unless, in addition, the field as a whole is extremely contracted.

Undoubtedly, however, in certain cases of this type the field shrinks after satisfactory reduction of pressure by an operation. Thus a field extending to 30° on the temporal side but only to less than a degree from the fixation area on the nasal side may become reduced to 10° to 15° or less. These fields tend to be horizontally oval and very steep-edged with fairly well preserved central vision, at least, at first. The usual form of contraction characteristic of glaucoma is replaced by a concentric restriction. It would appear that something of the nature of a progressive glial cicatricial process occurs in the nerve.

When central vision is found to be definitely depressed at an early stage, a comparatively rare occurrence, it is well to consider carefully the possibility of a complication, especially central choroido-retinitis.

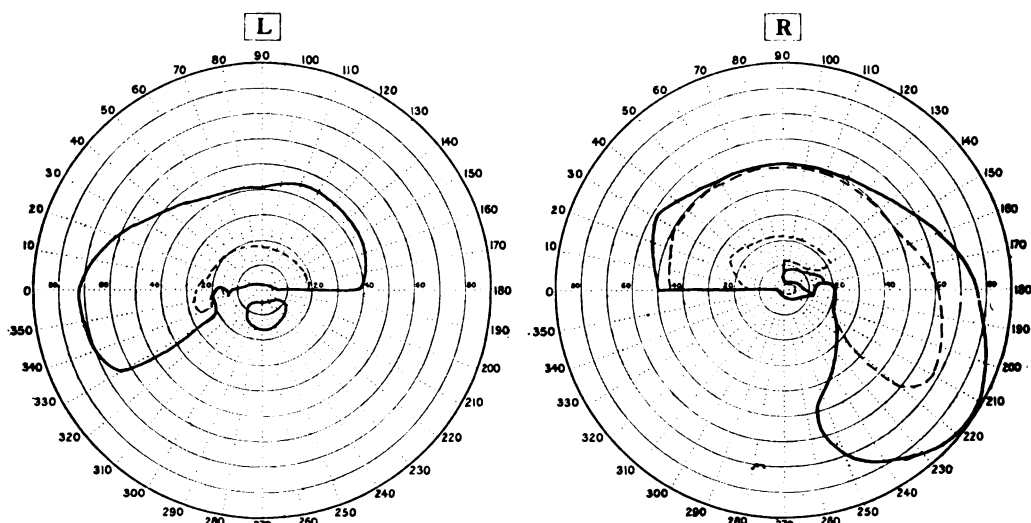


FIG. 87.—GLAUCOMA WITH CENTRAL SCOTOMA.

Retrobulbar neuritis (left) twelve years previously: L.V. = F. at 4 metres. Now nearly $\frac{6}{80}$. Field shows central defect. Objects $\frac{3}{30}$, $\frac{3}{30}$.

R. field shows typical features of glaucoma. Objects $\frac{3}{30}$, $\frac{3}{30}$, $\frac{2}{60}$. V. $\frac{6}{24}$. (M., 1924.)

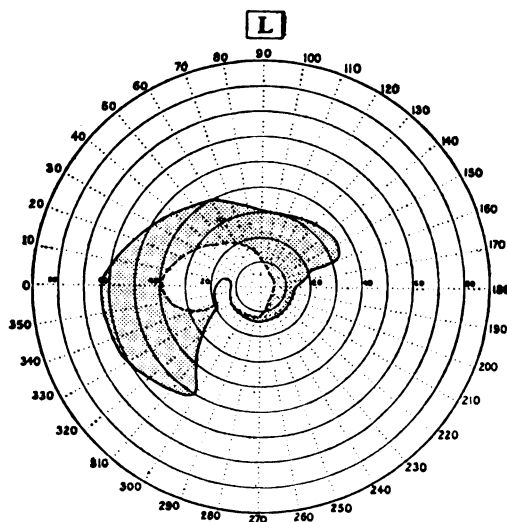


FIG. 88.—SECONDARY GLAUCOMA.

From a case of chronic cyclitis. (G., 1921.)

Objects $\frac{3}{30}$, $\frac{3}{30}$. V. $\frac{6}{12}$.

In secondary glaucoma the field changes are of the same type as in the primary form, but may be complicated by the existence of choroidal or other changes. The perimetric evidence shows that the field changes of glaucoma are due to the pressure, and is, therefore, against the view that a cavernous atrophy of the nerve head occurs as a primary factor in glaucoma.

CHAPTER IX

THE OPTIC NERVE

For our present purposes the following grouping may be adopted :—

- I. Intoxication and inflammation.
 - A. The toxic amblyopias.
 - B. The various forms of retrobulbar neuritis, descending neuritis, and some atrophies without obvious preceding swelling of the optic disc.
- II. Pressure.
- III. Pterocephalic * œdema (œdema due to increased intracranial pressure).
- IV. Vascular disease.
- V. Traumatism.

I

A. TOXIC AMBLYOPIA

The majority of optic nerve affections fall into the first group. The term toxic amblyopia is usually regarded as referring to conditions due to the action of exogenic poisons such as tobacco, although toxins produced within the body, as in diabetes, produce somewhat similar effects.

Acute and chronic forms may be distinguished depending on the action of the poison, the dose, and the duration of administration : it is evident that clinically certain poisons will be found associated with the former and others with the latter variety. The field changes consist of central defects, peripheral depression, or a combination of both, and to a certain extent the kind of defect produced is specifically related to the causal agent. Thus, while the majority of toxic substances, which affect the field of vision, produce central defects with little or no peripheral involvement, quinine and some others always cause peripheral loss, if sometimes central in addition. In this connection Uhthoff has pointed out that two groups of poisons may be recognised. The first group contains poisons which also tend to produce peripheral neuritis, and includes such substances as lead, carbon bisulphide, arsenic and others. Among endogenic poisons in the same group may be quoted as examples those which produce central scotoma in diabetes, and in that interesting condition neuritis peripherica gravidarum. This group is characterised by the production of central scotoma without or with very little involvement of the peripheral fields. In the second group the field changes take the form of peripheral and general depression, while peripheral neuritis is rare. The most important substance in this group is quinine ; others such as salicylic acid and *Filix mas* may be included. Uhthoff (418) observes that tobacco appears to be an exception as no association with peripheral neuritis had been established although the field defect is central.

* From *πλήρης*, "full to overflowing." As "Papilledema" and "choked disc" refer to ophthalmoscopic appearances which may occur in local optic nerve disease this term has been chosen to indicate œdema of the disc due to increased intracranial pressure. Compare Von Hippel's (444) use of the term "Stauungspapille."

It is also of interest to note that differences in the chemical constitution of toxic substances can modify their action as regards the visual field in important ways. For example, central scotoma is rare in poisoning by quinine, but occurs in connection with its close relative ethyl-hydro-cuprein, which is much more toxic to the visual neurones. Again, as has been pointed out by Schirmer, inorganic arsenic compounds produce central scotomata which tend to recover, while toxic organic arsenical preparations such as atoxyl produce peripheral depression advancing to complete blindness.

Such examples suggest the possibility of establishing some degree of correlation between the toxins and the field changes they produce, by the recognition of the characters of the defects, and show that perimetry has, in this respect, a definite role to play, although its value cannot yet be correctly estimated. The whole subject of the relationship between different toxic substances and their specific effects upon the visual functions forms a field full of interest and as yet but little explored.

The lesion produced by the toxin is probably in many instances, *e.g.*, quinine, in the retinal ganglion cells, in others possibly in the nerve fibres. There is no perimetric evidence of any primary incidence of the lesion at a higher level than the optic nerve, so that the suprachiasmal part of the visual pathway would appear to be almost immune to the initial manifestations of toxic and inflammatory disorders of the type which affect the sub-chiasmal part.

Nerve fibre bundle defects have never been found in toxic amblyopia, although they occur in retrobulbar neuritis. Although this may be regarded as negative evidence in favour of the site of interference being in the ganglion cells, much weight cannot be attached to it, as there is no doubt that toxins can affect the fibres in the nerve. It rather suggests that nerve fibre bundle defects arising in retrobulbar neuritis are due to causes not primarily toxic, but probably vascular.

**Group I. Bilateral Centrocaecal or Central Scotomata with little or no Involvement
of the Peripheral Field
Tobacco Amblyopia**

As an example of the scotomatous group, we may examine the amblyopia produced by tobacco as, owing to its frequency and gradual development, this is the most important and most easily studied form. Although often spoken of as central, the scotoma is typically centrocaecal in position, and is never central in the true sense, that is to say, pericentral. In shape it is horizontally oval though somewhat irregular in aggravated cases. Its characters are those of a conduction defect of moderate intensity with very gradually sloping edges, and it is thus most easily detected by a reduced stimulus such as a red or a small white test-object. At the time when the patient seeks advice the defect is always bilateral, and usually more developed in one field than in the other. The edge which overlaps the fixation point is always curved and never lies exactly along the vertical meridian, while its upper and lower boundaries curve temporally to the blind spot somewhat irregularly and usually asymmetrically. They do not follow the lines of the nerve fibres nor is a nasal step ever present. The

margins are very sloping and difficult to define exactly, and the defect for colour greatly exceeds that for white. A characteristic feature is the presence of one or two areas of greater intensity, or nuclei, within the scotoma. When single, the nucleus lies between the fixation point and the blind spot; when double, one nucleus is usually present just to the temporal side of the fixation point, and another near or at the nasal side of the blind spot. These areas lie on, or close to, the horizontal meridian and become connected as the defect increases, forming a horizontal core of the scotoma extending from the blind spot to the temporal edge of the fixation area.

If we trace the development of this scotoma we find that it begins by the appearance of a defect between the upper part of the blind spot and the fixation area on or near the

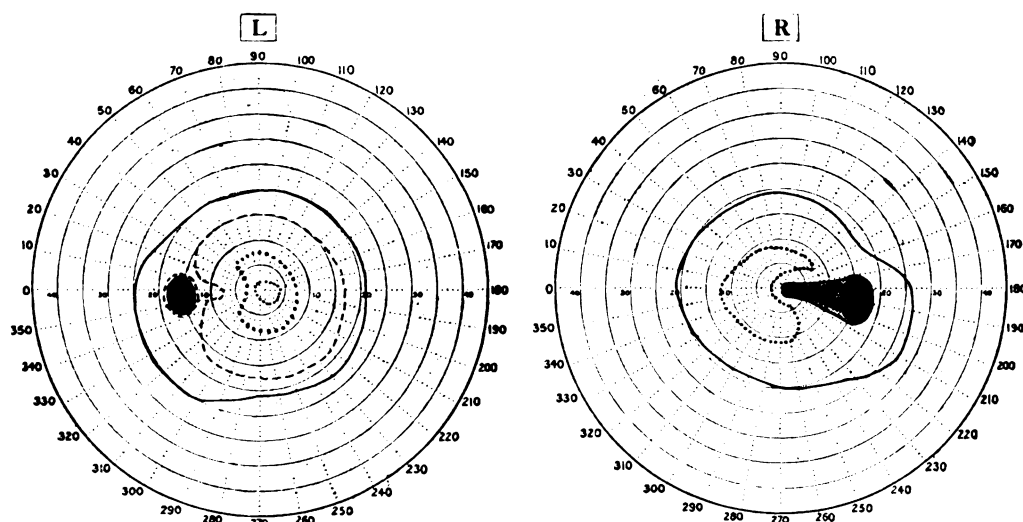


FIG. 89.—CENTRAL FIELDS FROM AN APPARENTLY UNILATERAL CASE OF TOBACCO AMBLYOPIA.

The right field shows typical changes; V. $\frac{6}{6}$. The left field shows a very early defect; V. $\frac{3}{6}$. The pericæcal amblyopic zone is intact and the blind spot is not enlarged.

Objects:—R. $\frac{2}{2000}$, $\frac{2}{1000}$ (blind spot and scotoma), $\frac{2}{2000}$, and $\frac{2}{1000}$ red (dotted lines); L. $\frac{2}{2000}$ (blind spot), $\frac{2}{2000}$, $\frac{2}{2000}$, and $\frac{2}{2000}$ red. (C. 1925.)

horizontal meridian, that is to say, the part which is found later as the nucleus is the first to appear. The defect may consist of a minute scotoma situated close to the nasal side of the blind spot or to the temporal side of the fixation area. There may be only one small scotoma between these two points. It is characteristic that these scotomata lie on the horizontal meridian and, thus, when they later become merged into a larger defect this may assume the form of a pointing index finger (Fig. 93 A). The scotoma does not develop as an outgrowth from the blind spot but as a paracæcal defect which at a later stage merges with the blind spot. At this stage vision is $\frac{6}{6}$ or thereabouts by Snellen's test, and the patient notices no defect or only a slight mistiness. In certain patients it may be extremely difficult or impossible to demonstrate the scotoma in its initial stage, although from the symptoms and history its existence is practically certain. This is, however, a very uncommon occurrence, and depends more on the patient's

G.P.

L

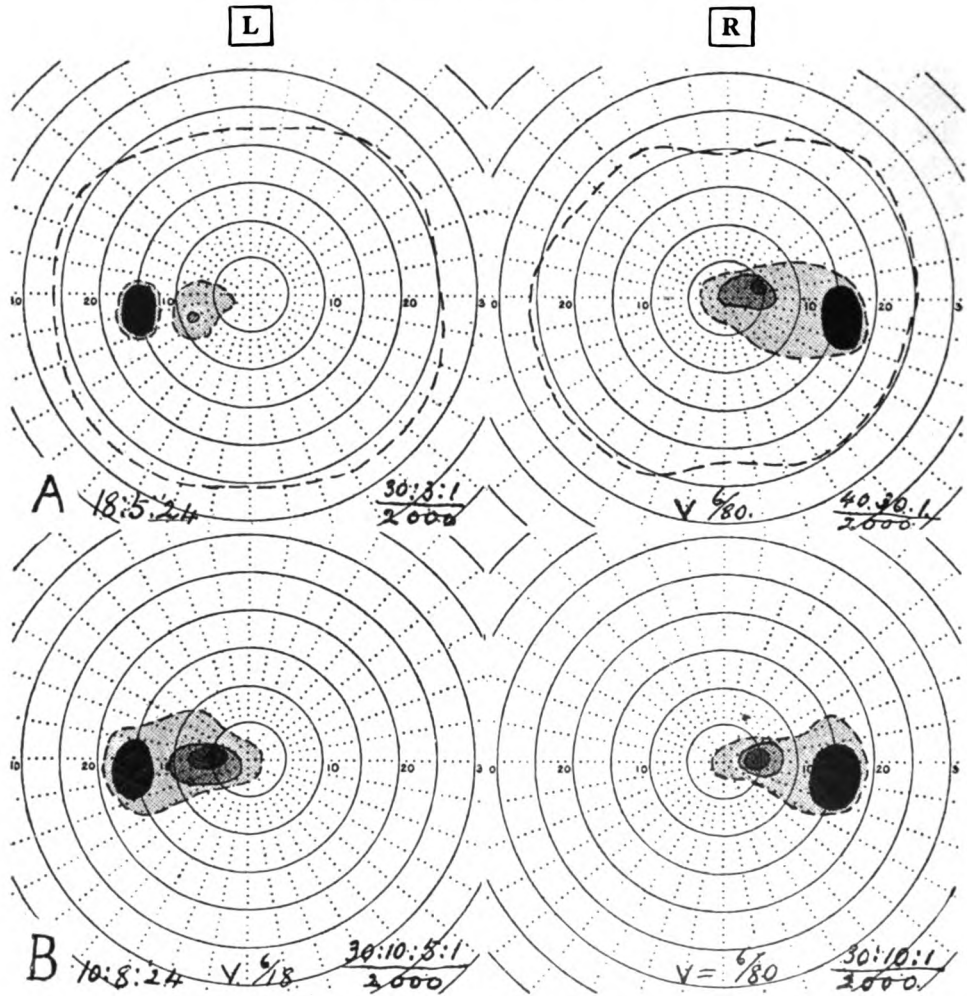
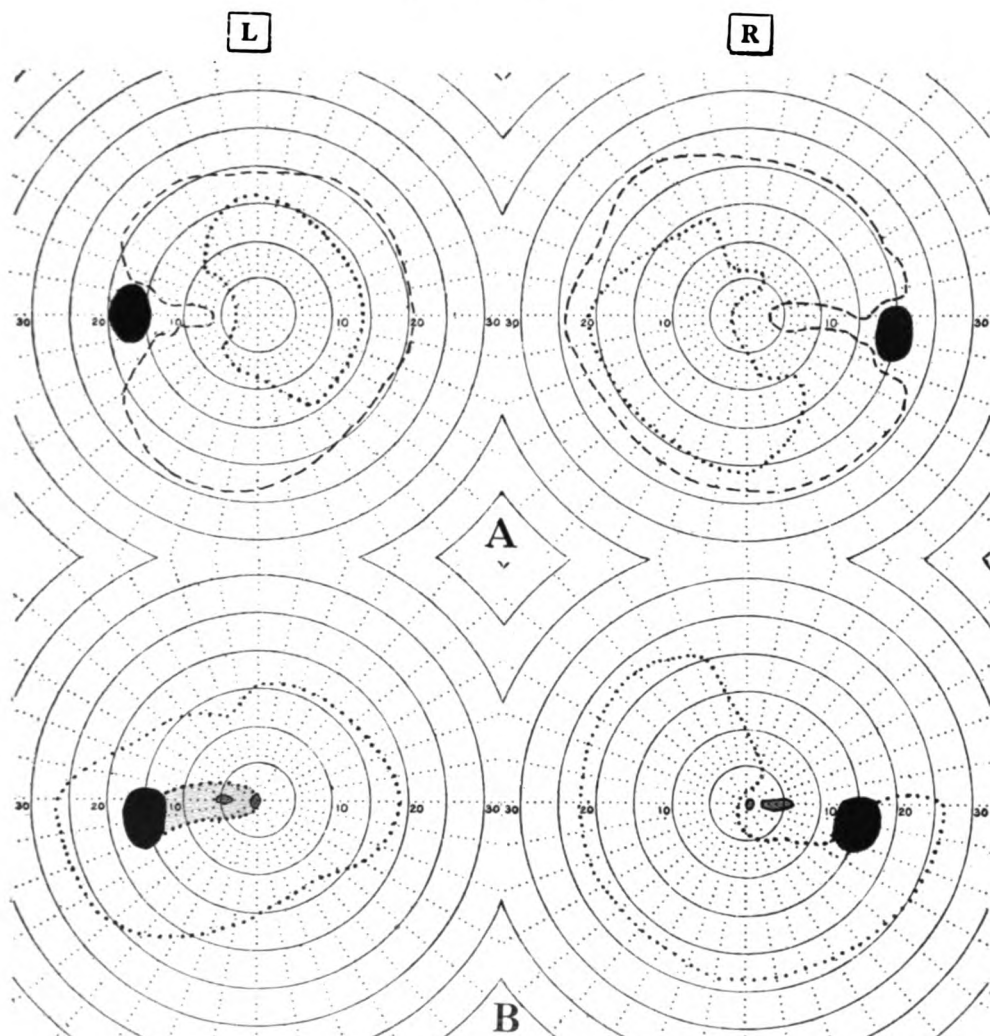


FIG. 90.—TOBACCO AMBLYOPIA.

- A. Shows an early stage, fixation area not yet invaded in left field. V. $\frac{6}{80}$. Defect well developed in right. No peripheral depression for 2000 white.
- B. Same after reduction of tobacco to half an ounce weekly for three months. Increase of defect in left field, denser nucleus in right field with slight contraction of scotoma near fixation area. (McG., 1924.)

anxiety to give positive responses than on the faintness of the defect, for, almost without exception, when symptoms occur, the scotoma is demonstrable. The early stage before the fixation area is involved can only be investigated in cases in which one eye is affected far in advance of the other, and it is not often that an opportunity of examining this stage occurs. This disparity in the development of the defect in the two fields indicates that tobacco amblyopia may begin unilaterally, although patients never present themselves until both fields are affected.

The scotoma now merges into the pericæcal amblyopic zone and extends towards the fixation point as a tongue-shaped projection from the blind spot. The defect is larger for colour than for white, and the patient begins to notice his sight failing when the edge of the colour defect begins to invade the fixation area. A second nucleus near



TOBACCO AMBLYOPIA.

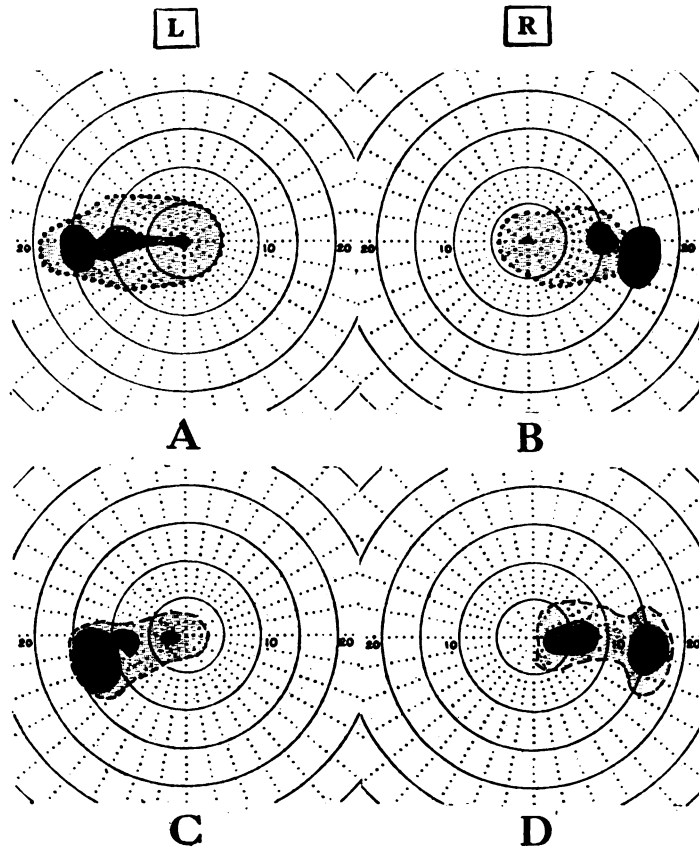
FIG. 91.—MODERATELY EARLY STAGES IN TWO CASES.

- A. Objects $3\frac{1}{2}$ (blind spot), $2\frac{1}{2}$ red. Note defect extending centralwards from blind spot, and depression for red not involving fixation area in the left field, but involving it in the right. R.V. $\frac{6}{38}$; L.V. $\frac{6}{38}$. (L., 1922.)
- B. In the left field a centrocaecal defect for red $2\frac{3}{4}$ with dense areas in which $2\frac{3}{4}$ white is not seen. The field for red $2\frac{3}{4}$ shows an indentation in the upper temporal quadrant, but is not yet broken through. V. $\frac{6}{12}$. The right field shows characteristic breaking through for red $2\frac{3}{4}$, with two small areas of intensity. V. $\frac{6}{12}$. (H., 1923.)

Dotted lines indicate fields for red.

the fixation point may now become demonstrable if it has not already appeared. Although the onset is in reality gradual, the patient sometimes states that his vision failed or became much worse suddenly. This statement is usually made in relation to reading and indicates the rapid severe impairment of a small number of macular nerve elements when the temporo-central nucleus develops. The centrocaecal scotoma is now definite for colour but may require an angle as small as $\frac{1}{2000}$ for demonstration with white, while the nuclei may be very much more intense. At this stage the peripheral field for white

L 2



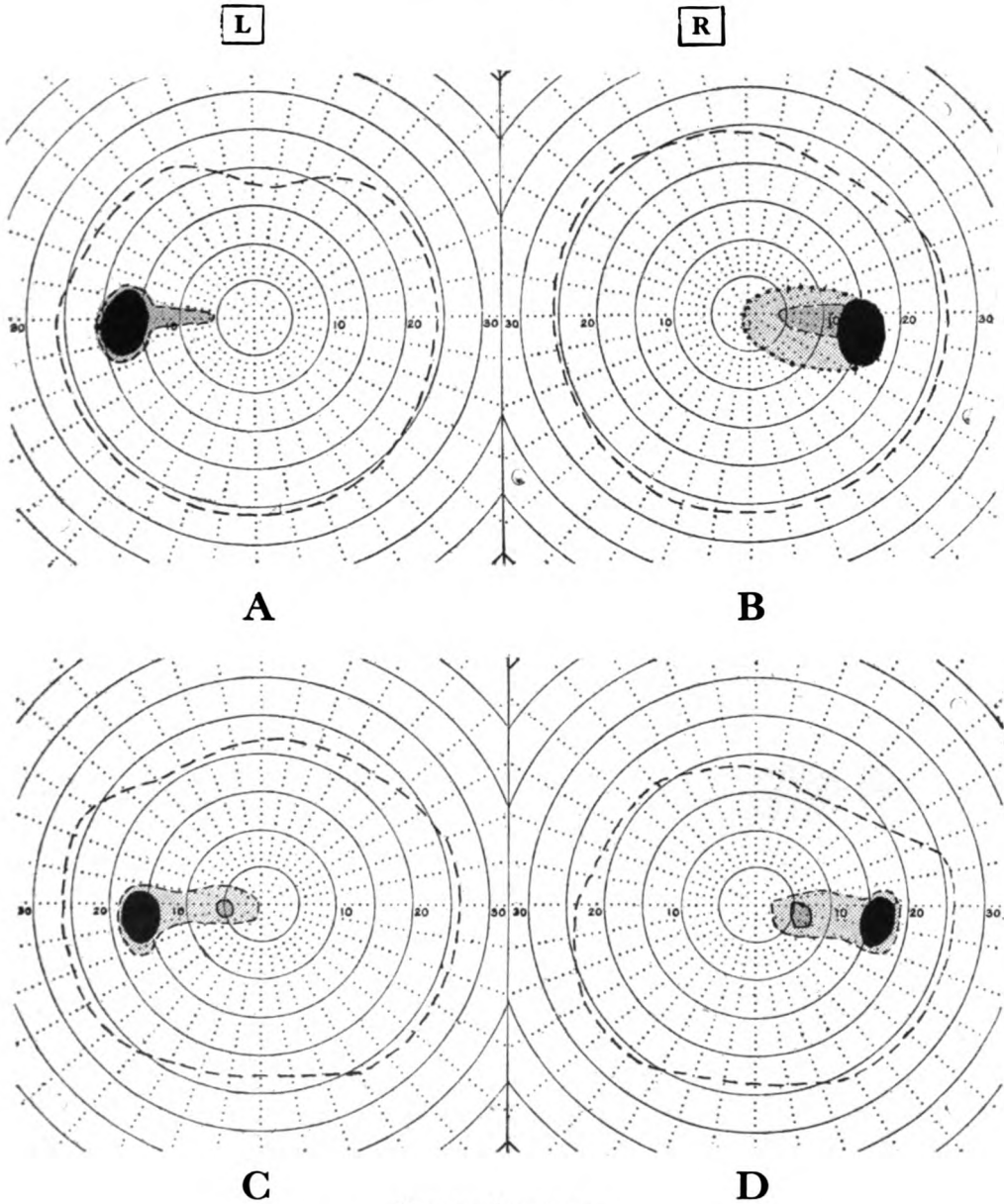
TOBACCO AMBLYOPIA.

FIG. 92.—DIFFERENT FORMS OF THE AREAS OF INTENSITY.

A central core. B and C, separate intense areas in the most common positions. D, single intense area. Objects. A and B, $\frac{2}{3}$ red, $\frac{1}{3}$ white (F., 1912); C and D, $\frac{2}{3}$ red, $\frac{1}{3}$ white, and $\frac{1}{3}$ white (W., 1922).

and colour will be found intact for large visual angles, but if examined on the screen the colour field within the 30° circle will often be found impaired in the upper temporal, or in both temporal quadrants, in addition to the oval area of the scotoma, showing that the involvement of the retina is by no means confined to the papillo-macular area. Central vision varies from $\frac{6}{60}$ or even $\frac{6}{6}$ to about $\frac{6}{36}$ according to the relation of the scotoma to the fixation area.

As development proceeds the scotoma enlarges, its central core increases and becomes apparently absolute in parts, and the fixation area is well overlapped, so that central vision falls to $\frac{6}{60}$ or less, although the edge of the scotoma rarely extends to more than 5° or 6° to the nasal side of the vertical meridian. A common feature is a vertical constriction of the outline of the scotoma near the blind spot so that it appears to be joined to the blind spot by a *col* or neck. The colour field becomes severely affected, the scotoma breaks through above, usually in the upper temporal quadrant, and then the lower quadrant fails, so that the field for red is reduced to a crescentic area on the nasal side. In severe cases all perception of red may be lost according to Grœnouw,



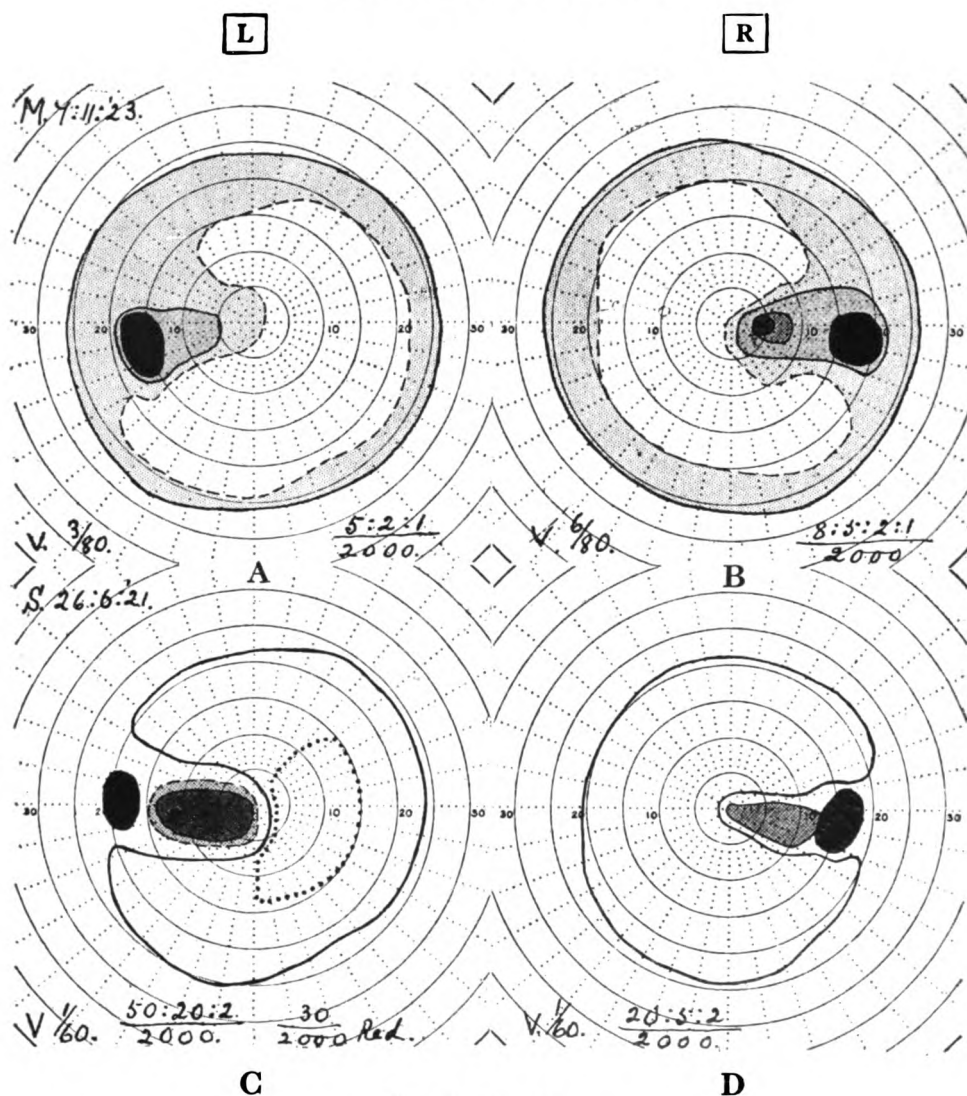
TOBACCO AMBLYOPIA.

FIG. 93.—EARLY STAGES WITH MODERATE REDUCTION OF VISION.

Note absence of depression of central field for white ($\frac{2}{2000}$ isopter normal). Vision: A, B, C, $\frac{3}{36}$; D, $\frac{6}{12}$. (B., 1922; Mc., 1924.)

but I have not found this if large enough objects are used. Similarly, the temporal field for a small visual angle for white, $\frac{2}{2000}$ for example, may be lost. Harman found slight peripheral contraction for small test-objects indicating some general depression of the whole field. Complete blindness appears never to ensue even in very aggravated cases although the use of tobacco is continued.

During recovery the defect retraces its steps, and, in a general way, those features

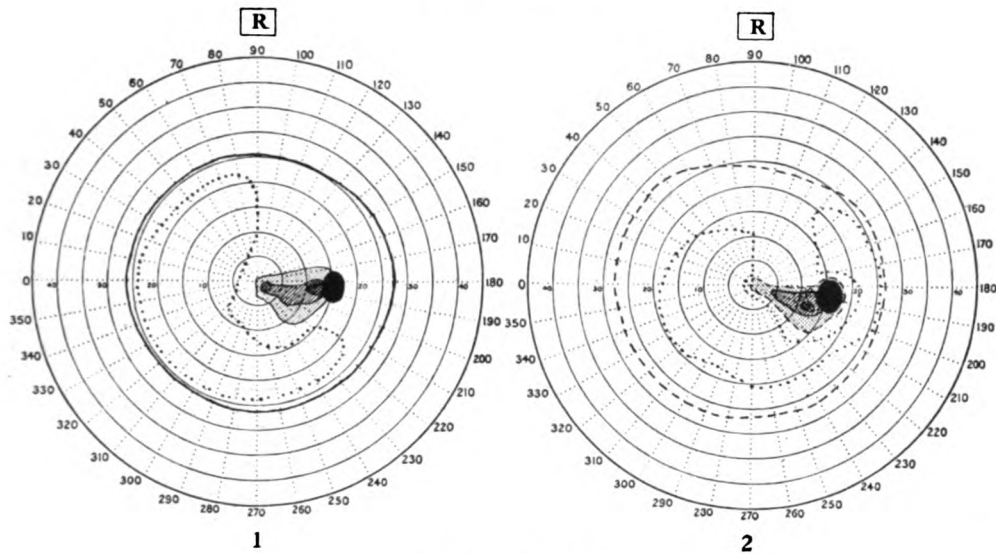


TOBACCO AMBLYOPIA.

FIG. 94.—MORE ADVANCED STAGE.

In A and B, central field for $\frac{1}{2000}$ reduced, isopter for $\frac{2}{2000}$ in position of that for $\frac{1}{2000}$. Scotoma more intense. In C, note very intense scotoma with field for $\frac{30}{2000}$ red reduced to a nasal patch. Central vision poor.

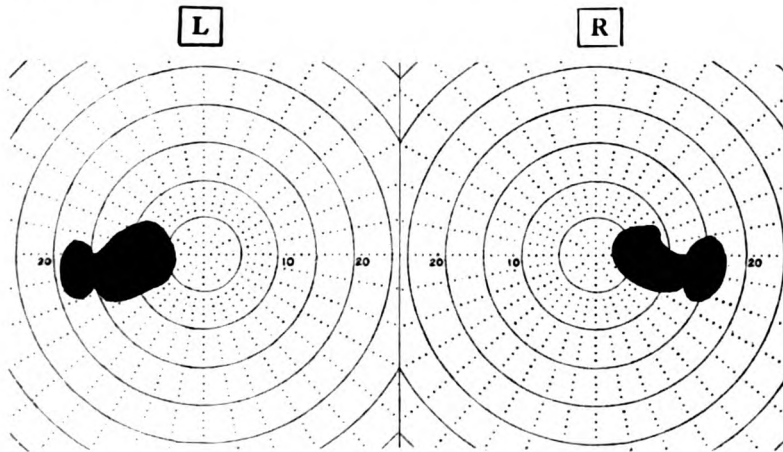
which appear last disappear first. In cases which are obscure or doubtful at first owing to the severity of the visual depression, characteristic features emerge as recovery proceeds. The colour field creeps round into the temporal field, the scotoma becomes smaller, more defined and more steep-edged, the core or nuclei remaining longest. In very severe cases quite good central vision may be restored with large absolute scotomata projecting nasally from each blind spot, but not reaching the fixation area. Central vision may return very rapidly or even almost suddenly after a period of slow and disappointing progress, a feature which may be explained by the rapid recovery of some foveal elements. Recovery does not always closely follow abstinence



TOBACCO AMBLYOPIA.

FIG. 95.—RECOVERY.

1. Central field of the right eye in a case of tobacco amblyopia. V. $\frac{6}{80}$. The field for red $\frac{20}{200}$ shows a large upper temporal effect. Note the nucleus and nucleoli in the scotoma.
2. The same after abstinence from tobacco for a fortnight. V. $\frac{6}{35}$. The field for red is extending upwards into the upper temporal quadrant. The scotoma and its nucleus are retreating towards the blind spot. The field for white which previously required a 2-mm. object (continuous line), can now be delimited by a 1-mm. object (broken line.) (D., 1923.)



TOBACCO AMBLYOPIA.

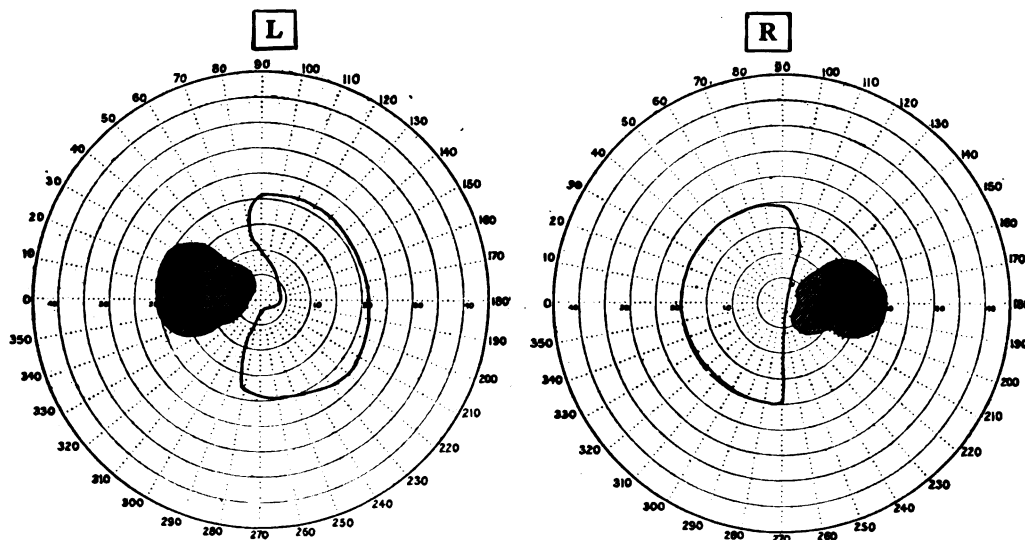
FIG. 96.—PARACENTRAL DEFECTS AFTER CONSIDERABLE RECOVERY OF CENTRAL VISION IN A SEVERE CASE: CONDITION THREE AND A HALF YEARS AFTER ONSET.

Dense practically absolute scotomata, steep-edged and not encroaching on fixation area, though relative defect for red extends to fixation area. R.V. $\frac{6}{34} +$; L.V. $\frac{6}{12}$ part. In stage of advance vision with such a dense scotoma is much worse as the scotoma is surrounded by an amblyopic zone and is not so steep-edged. Objects R. $\frac{20}{200}$, L. $\frac{40}{200}$. (M., 1923.)

from tobacco; the vision may remain stationary or may decline during the first four or six weeks owing to a more extensive invasion of the fixation area. This corresponds to the history so often obtained, that tobacco had been stopped or cut down for some time before advice was sought. Snellen's test is inadequate in these cases for, although

central vision may appear stationary when examined in this way, the patient often says that he sees better and the campimeter shows a recession of the defect.

Interpretation.—It is evident that the study of the characters of the field defects gives no positive indication as to the site of interference, which might be either retinal or neural. The absence of nerve fibre bundle or zonular defects may be regarded as negative evidence in favour of the site being in the retinal ganglion cells. The peculiar distribution of the scotoma, almost entirely in the area of the crossed fibres, is common in retrobulbar neuritis and corresponds to no special structural or vascular arrangement in either retina or nerve as far as is known, nor to any functional quality, the macular elements being involved very unequally and severely only in aggravated cases. The



TOBACCO AMBLYOPIA.

FIG. 97.—STATE OF CENTRAL FIELDS THREE YEARS AFTER ONSET IN A SEVERE CASE IN WHICH VERY LITTLE RECOVERY HAS TAKEN PLACE.

Condition now practically stationary. Objects 2000° , 2000° , 2000° . The vertical elongation of the blind spots (2000°) is probably partly due to bad fixation. R.V. = 2000° . L.V. = 2000° . (S., 1923.)

evidence is strongly against the view that those elements whose function is most highly developed are more easily affected. We must remain contented with the assumption that certain nerve elements have a special affinity for the poison of tobacco, and that they are affected in the order of their susceptibility. The pronounced tendency of the nuclei and of the scotoma itself to lie along the horizontal meridian suggests that the susceptibility may be based on nutritional and ultimately on anatomical factors (see Behr 28). This view is supported by the fact that in tobacco poisoning no other type of field change is produced. Increase of the field defects after the disuse of tobacco indicates that the poison is to a certain extent cumulative in its action, while the failure to culminate in complete blindness, although tobacco is continued, suggests the attainment of a certain degree of tolerance. Although the worst cases are usually related to the most excessive use of strong tobacco, the great variation of consumption in relation to the field changes in different cases shows that idiosyncrasy and resistance are important

factors. The presence of disproportion, shown by the excess of the defect for red over that for white indicates a diffuse lesion in which the conductivity of many nerve elements is partially impaired, but of only few severely. Lastly, the evolution during recovery in severe cases of a smaller steep-edged dense scotoma shows that the less affected elements are recovering, while those which were badly damaged or killed remain functionless.

Diagnosis.—The perimetric diagnosis of tobacco amblyopia depends upon the recognition of the special characters of the defects. The diffuse nature of the scotoma and its sloping ill-defined margins are valuable signs. Errors may be made when the mere presence of the scotoma and perhaps its size, shape, and position alone are taken into consideration. One source of difficulty is the detection of the nuclei only, owing to omission to use an adequate range of visual angles when testing on the screen. The characters described, regarded collectively, do not appear to occur in any other form of amblyopia. They are very constant, so much so that atypical defects may justifiably be regarded with extreme suspicion, almost amounting to certainty, that they are not due to tobacco, while if not only the field defects but the whole clinical picture, and particularly the history, be considered, misdiagnosis should be easily avoidable, especially in ordinary cases of moderate severity. Even in females, who may hesitate to admit the use of tobacco, the demonstration of typical defects enables a diagnosis to be made with confidence. Very severe cases with extreme reduction of vision and bad fixation may offer difficulty, especially if anomalies are present in other parts of the clinical picture.

According to Lillie the size and shape of scotomata due to tobacco and to other causes depend upon the stage the condition has reached and no special form is associated with tobacco. This may be true as far as these two characters alone are concerned but does not affect the diagnosis when all the characters are taken into consideration.

In some severe cases eccentric fixation is present, which gives rise to findings which may prove misleading if their nature is not appreciated. The patient looks with one eye to the nasal side of the fixation point, so that the scotoma is moved into the nasal field and the defects appear homonymous instead of bitemporal. As the whole field is moved laterally, the defect for red in the upper temporal or upper part of the field comes to lie upwards or upwards and nasally. I have never found this condition in connection with both eyes, and in the three or four cases I have observed the false fixation has always been in the left eye. It is easily detected because upon the screen the blind spot is found much too near the fixation point, and the defect in the binocular field does not correspond to those found uniocularly, as it would were it really of homonymous hemianopic type. Occasionally the displacement is upwards or downwards. It may be that the dim appearance of the fixation object induces a state of groping and a tendency to extrafoveal fixation. A possible explanation is that the displacement of fixation may be due to the presence of a specially dense nucleus towards the central end of the scotoma overlapping the fixation area. The patient, doing his best to keep the fixation object in sight, apparently fixes with a part of the retina between the optic disc and the fovea which corresponds to the temporal border of the nucleus, instead of, as is usual, fixing with the fovea or with a retinal area slightly to the lateral side of the fovea, corresponding in the field to the nasal border of the nucleus. That this

is the case is indicated by the fact that a similar displacement, though only a couple of degrees in extent, sometimes occurs when quite a small but very dense nucleus lies immediately on the temporal side of the fixation area. During the screen examination the nucleus moves to the nasal side of the fixation point, and then the blind spot will be found to have moved also, while the patient explains that he has difficulty in keeping the fixation object in sight.

A rough test may be made by comparing the vision for red a few degrees to the nasal and temporal sides of the fixation area, selecting both object and distance in accordance with the requirements of the individual case. Should there be doubt, the scotoma must be carefully analysed and the whole field examined. Occasionally the defect is much more advanced in one field than in the other, and a careful screen test of the apparently normal field is necessary to demonstrate the defect, which may consist merely of a tongue projecting inwards from the blind spot. Although, theoretically, the onset is not likely to be simultaneous in the two fields, no exhaustively examined unilateral case has been recorded, and both fields appear always to be affected by the time any visual change is noted by the patient, that is to say, by the time that invasion of the fixation area of one side has commenced.

If the intense part of the defect extends considerably to the nasal side of the fixation point or is localised above or below it, or if the scotoma is truly pericentral in either field, or if the defects are of different types in the two fields, or sharply defined with steep edges and high and relatively uniform intensity, other forms of disease must be carefully excluded, and it is well to remember that excessive use of tobacco may exist along with some other condition to which the visual symptoms may really be due. Progressive increase in the defects, not mere failure of central visual acuity to improve, continuing for some weeks after cessation of tobacco, suggests a complication which is certainly present if peripheral contraction for an angle as large as $\frac{5}{330}$ occurs.

Alternative diagnoses are, therefore, confined to other forms of bilateral scotomatous amblyopia, in which the scotomata are centrocæcal and symmetrical. The defects of retrobulbar neuritis are denser, more polymorphic and less regular. A useful differentiating feature is the more definite limitation of the edge of the defect and its greater intensity in the early stages in such cases. Uhthoff has pointed out that the defect for blue is practically co-extensive with that for red in retrobulbar neuritis, but much smaller and confined to the middle of the scotoma in tobacco poisoning, an observation which indicates the sloping margins and the diffuse relative nature of the tobacco scotoma as against the denser and more steep-edged defect of retrobulbar neuritis.

The fact that the scotoma is easily elicited by a colour test such as red or green does not mean that red and green are specially affected by tobacco, or, for that matter, by any other toxic agent, that is to say, that depression of perception of red and green is specially diagnostic of a toxic cause. Prominence of the colour defect merely indicates the relative nature of the field change. The colour test is convenient, since it enables a larger object, covering a larger retinal area, to be used, and other methods of attaining the same object, such as reducing the illumination, give similar results.

In cases of general disease, such as diabetes, in smokers we shall be correct as a

rule in attributing the amblyopia to the tobacco if the scotoma is typical, and, in any case, the causation of scotomata by endogenic toxins in diabetes and other diseases, although extremely rare, must be borne in mind. Depression of health due to disease, especially high blood pressure, injury or even mental shock is often responsible for the onset of tobacco amblyopia in smokers, and on this basis it sometimes follows operation for cataract. In disseminated sclerosis, bilateral relative scotomata have been reported, but the characters, especially the behaviour, of such defects are always different from those of tobacco amblyopia. Similarly Leber's disease, saturnism, and retrobulbar neuritis due to nasal sinus inflammation, will not be mistaken for tobacco poisoning if all the characters are taken into consideration, to say nothing of the history, the ophthalmoscopic signs, and other clinical features. The centrocæcal scotoma of hypophysial tumour has been frequently mistaken for tobacco amblyopia, and the resemblance is greatest in aggravated cases in which the loss of one or both upper temporal quadrants of the fields for red produces an appearance resembling a bitemporal hemianopia (Figs. 91, 97). In cases of chiasmal interference from a slowly acting cause, such as tumour, the defects are more sharply demarcated, and commencing temporal hemianopia for white is usually present, while central vision is not so much depressed as in severe tobacco poisoning, unless a definite dense defect involving the fixation area is present. The defects are more easily analysed with white test-objects, the disproportionate loss of colour vision is not so pronounced, the characteristic core is not present, and the special quadrantic features of the chiasmal scotoma are usually easily demonstrable.

When tobacco amblyopia occurs as a complication of nervous disease such as hemianopia or tabetic atrophy, the diagnosis may be difficult, but can usually be cleared up by careful examination. The depression of health associated with these conditions may bring on the amblyopia, and caution should be exercised before attributing the bad central vision to the hemianopia or the tabes.

Prognosis.—In moderately severe cases in which the scotoma exhibits sloping edges and separate nuclei or a narrow central core, with little impairment of the temporal field for red, good vision is usually restored in a few months if tobacco is stopped. Improvement of vision may be due to improvement in the patient's general health even if the tobacco is not stopped. When there is an extensive dense steep-edged scotoma for white with severe impairment of colour vision in the central field, great depression of central vision and some pallor of the optic discs, recovery is always prolonged, and usually imperfect. Complete blindness, however, does not occur. Failure of central vision to improve, or even a decline of central vision during the first four or six weeks after tobacco is stopped does not indicate a modification either of prognosis or diagnosis. Should slow progress cause disappointment and anxiety the screen test is valuable, as indications of improvement, not noticeable by the patient, may be discovered, and if such are found he may be encouraged to hope for the ultimate recovery of useful vision.

Alcohol Amblyopia

Neither ethyl alcohol nor methyl alcohol can be credited with special toxic effects upon the visual nerve path. The field changes described above under tobacco amblyopia

are commonly referred to in the literature as due to tobacco plus alcohol, or even to alcohol chiefly or alone. While it would be out of place to discuss this question here, it may be remarked that, although spirit drinking is no less prevalent than in other countries, amblyopia traceable to alcohol, apart from tobacco, is almost unknown in British hospital clinics,* and the experience of British ophthalmologists does not support the view that alcohol is a potent factor. Convincing evidence is accumulating that impurities, such as wood spirit or fusel oil, which are violently toxic, are the real cause of the visual disturbances and that the prevalence of so-called alcohol amblyopia in any country (on which differences of opinion are based) is an indication not of the amount of alcohol consumed, but of its purity.† Under the heading of alcoholic amblyopia, reference will therefore be made to the field changes produced by these toxic substances which may be consumed or inhaled in the form of commercial wood spirit or alcoholic drinks adulterated with unpurified spirits.

The clinical picture as described by Uhthoff, Goldflam and others, is that of an acute intoxication. Usually two or three days after ingestion, or earlier in severe cases, the visual loss commences, and complete or nearly complete blindness rapidly ensues. Both eyes are always affected though often unequally. After several days, or even weeks, according to the severity of the case, vision begins to return at the field periphery, and a large absolute defect, broken through at one side or the other, becomes demonstrable. This breaking through may give the field an irregular crescentic or sometimes pseudo-hemianopic appearance. Later the periphery may recover more or less all round, leaving a central scotoma.

The defect is pericentral rather than paracentral or centrocaecal, though it often includes the blind spot and the intensity is usually high. According to the severity of the case, all degrees of size, intensity, and permanence may result. In the worse cases little or no recovery may take place, in the milder ones normal vision may be restored. This stage is unfortunately frequently succeeded by a second and permanent loss of vision associated with pallor of the optic disc. The field becomes depressed and contracted: Goldflam noted a return to blindness in a case which had reached normal vision. Only exceptionally does good vision return and remain when the field changes have been severe to begin with.

The field changes indicate the action of a violent poison on the retinal cells and optic nerve with special selectivity for the central elements. The ultimate deterioration of vision following primary recovery is probably due to a cicatricial process in the nerve, depending on the violence of the reaction, leading to secondary atrophy.

The diagnosis depends upon the clinical picture as a whole, which always suggests this form of poisoning: the prognosis should be guarded at first, even although the field defects have greatly improved, as secondary atrophy may supervene. The fields should, therefore, be watched for some time after apparent recovery.

When the poison is ingested in smaller doses over a longer period a chronic form

* "... no authenticated instance . . ." Dep. Committee on the Causes and Prevention of Blindness. Ministry of Health, 1922.

† Hamäläinen and Teräskeli. *Acta Ophthal.*, VI., 260, 1928.

of this intoxication occurs. Blegvad and Roenne found that during the 1914-18 war, owing to the dearth of the usual form of potable spirit in Denmark, substitutes were consumed, and a severe form of toxic amblyopia arose previously practically unknown in that country. Central vision was reduced to counting fingers in 80 per cent., and the optic disc was pale in 20 per cent. of the eyes examined. The field periphery was always normal (with $\frac{10}{300}$), while red and green were in many eyes not recognised anywhere. Every case showed a central scotoma for red and green, and in 50 per cent. for blue also. A third of the fields showed central or paracentral scotomata also for white, and breaking through of the defect above was also noted. The prognosis is worse than in the ordinary form of toxic amblyopia. The description given is not sufficiently detailed to enable all the characters of the defect to be assessed, but the field changes appear to differ from those of tobacco amblyopia in their course—they appear a few months or less after the drinking begins—and severity. The scotoma is described as central, and appears to be much denser ($\frac{10}{300}$ white not seen in the scotoma in half the cases) than even in the severe tobacco cases, and is therefore unlike the scotoma of tobacco amblyopia in this country.

Whether these patients were also smokers is not stated, but it is possible that in countries in which impure alcohol is commonly consumed a chronic form of alcohol or tobacco-alcohol amblyopia may be produced.

Other forms of scotomatous toxic amblyopia are uncommon. Their rarity and their usual acute development and course have prevented the elicitation and study of the characters of the scotomata to the extent which has been possible in the case of tobacco. The following notes are based on the publications of Uhthoff, de Schweinitz, and Wilbrand and Saenger.

Lead

The scotoma is bilateral, pericentral, roughly circular, and often absolute, though it may be relative. The field periphery is unaffected, but the colour fields may show breaking through. The onset is usually gradual, sometimes relatively rapid, and the field changes resemble those due to the common type of retrobulbar neuritis or to an active poison such as bisulphide of carbon rather than those of the tobacco amblyopia type. Complete blindness is an uncommon result (10 per cent., Uhthoff). This is the true toxic amblyopia of lead, due to a lesion in the optic nerve; other field changes also occur which depend upon the action of lead upon the blood-vessels or kidneys. The progressive depression and contraction of the field following papilloedema, and often leading to total blindness, may thus be explained as a secondary effect due to increased intracranial pressure caused by lead; similarly, the hemianopias and sudden bilateral amaurosis are indirect rather than direct results.

Carbon Bisulphide

Bilateral central scotomata are produced which vary in size and intensity according to the severity of the intoxication. In position the defect is usually more or less pericentral, though it often extends more to the temporal than to the nasal side of the fixation point, and, in size, it may vary from a minute area to an extensive scotoma of some 40°

in diameter. The intensity may be very slight, so that small test-objects are required, or the defect may be absolute in its central part with a surrounding amblyopic zone which may break through to the periphery. As in other conduction defects, red-green blindness is a prominent feature. The field as a whole may be depressed and contracted. The onset is usually gradual, but occasionally acute.

The scotoma differs in shape and intensity from that of tobacco, with which it is most likely to be confused, and the presence of more pronounced peripheral field changes aids the diagnosis. If the patient can be removed from exposure to the poison, the prognosis is good in the milder cases, and in the more severe some improvement usually follows.

Similar scotomata occur in connection with iodoform poisoning, and rarely from arsenic and some other substances, also in beriberi and other forms of avitaminosis and nutritional deficiency. As regards these, distinctive individual perimetric pictures have not yet been established.

A special form of nutritional neuropathy has become prominent in connection with World War II, and more especially in connection with prisoners of war. It is characterised by small central, sometimes centrocaecal, scotomata with occasional peripheral field defects and is associated with other nervous symptoms. There is no specific perimetric feature characteristic of this disease, which is not well understood.*

Group II. Bilateral Depression of the Fields Without or With Involvement of the Central Area

Quinine

In the second group of toxic amblyopias, *quinine* poisoning occupies the most important position. In mild cases a temporary dimness of vision may occur; when the poisoning is more severe rapid and complete blindness results. Peripheral vision is lost first and central vision finally. Minute central steep-edged fields with orientation difficulty may be present at a very early stage. After a few hours or days, sometimes weeks, vision begins to return, recovering centrally much more rapidly than peripherally, and the fields show depression, especially peripherally, and contraction. The light sense is greatly reduced. At this stage the edge of the field is extremely steep, and disproportion between the fields for colour and white is present. Though often more or less reduced, central vision by Snellen's types may be nearly or quite restored, while the peripheral field remains depressed and constricted. The loss of field may be extreme, the part remaining extending only to a few degrees round the fixation point; in such cases the patients exhibit great orientation difficulty.

In shape the outline of the field is often horizontally extended and vertically compressed, forming a somewhat horizontal roughly elliptical figure. This is due to an apparently greater tendency of the temporal and nasal parts than of the upper and lower to recover. The restoration may be irregular, some peripheral parts recovering, while other portions remain blind. In this way various forms of sector-like defect may be

* Spillane, J. D., and Scott, G. I. *Lancet*, 1945, 2, 261.

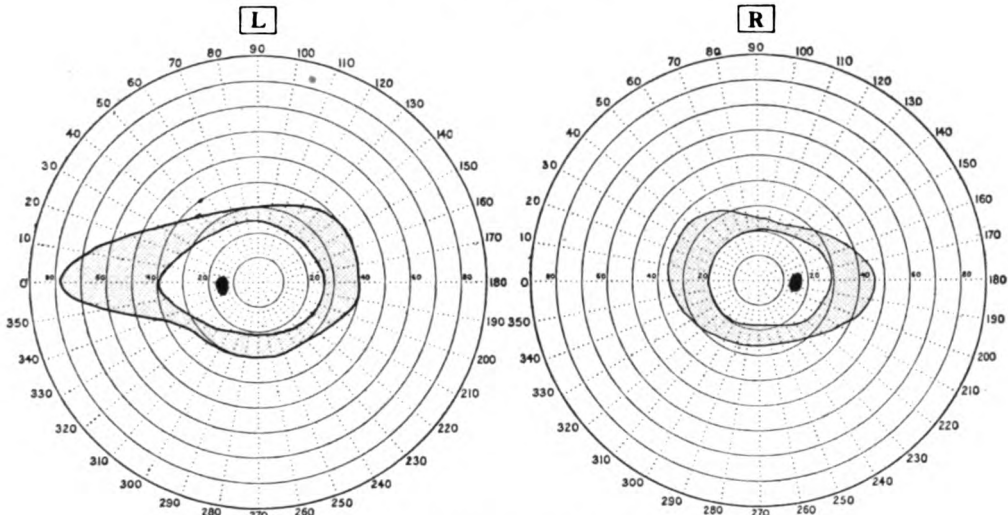


FIG. 98.—QUININE AMBLYOPIA; SEVEN MONTHS AFTER ONSET.

Objects $\frac{10}{336}$ and $\frac{60}{336}$. R.V., L.V. = $\frac{5}{6}$. Great peripheral with relatively little central depression. Red $\frac{20}{336}$ was recognised out to 15° to 20° from fixation point, and central colour vision was good (absence of disproportion) indicating improbability of further recovery. (A. B., 1916.)

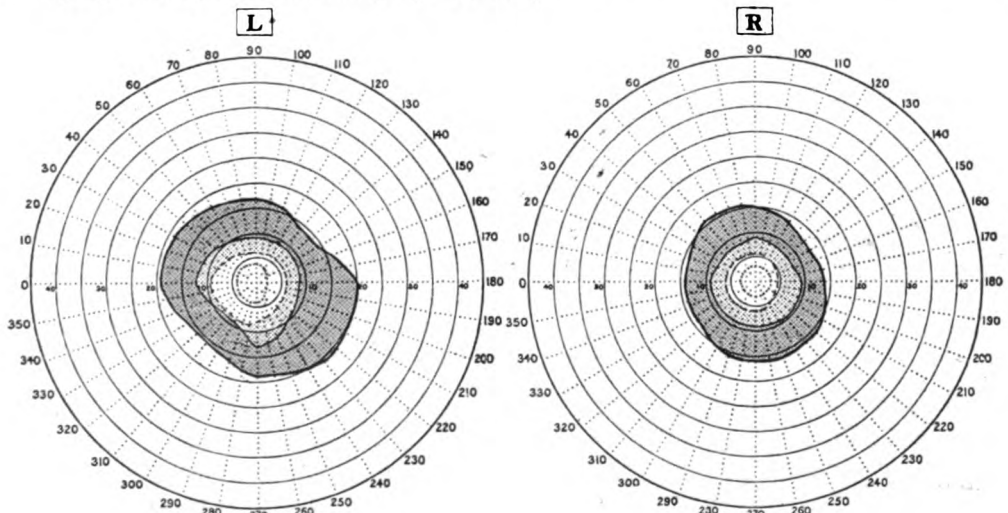


FIG. 99.—QUININE AMBLYOPIA. TEN DAYS AFTER POISONING, NINE DAYS AFTER COMMENCEMENT OF RECOVERY.

Test-objects $\frac{1000}{1000}$, $\frac{500}{1000}$, $\frac{1}{1000}$; dotted circle shows field for $\frac{3}{1000}$ red and blue. V. = $\frac{5}{6}$, each eye. Note the great steepness of edge of central field, $\frac{60}{1000}$ was seen only to 15° in the right field and to 20° in the left. There was also some peripheral vision for large objects, but orientation was very poor. (P. L., 1925.)

produced, and when the lost areas are symmetrical in the two fields, a superficial resemblance to hemianopia may arise. In other cases a more regular concentric contraction leading to an approximately circular field occurs. Much depends on the care with which the fields are taken as, especially in the earlier stages of recovery, discrepant and inconsistent responses are apt to be obtained, and stringent precautions to check the patient's replies are necessary.

Several months may elapse before the fields become stationary, and in mild or favourable cases they may recover almost completely, but some peripheral depression

persists ; in more severe cases a more or less high degree of depression and contraction remains permanently. Central scotoma has been recorded, but is very rare, and possibly really a complication rather than a true manifestation of quinine poisoning. Colour disproportion disappears as the condition becomes stationary.

Interpretation.—Differing views have been expressed as to whether the field changes are due to intoxication of the ganglion cells or to an ischæmia resulting from contraction of the retinal artery. There seems to be little doubt that the poison acts directly on the ganglion cells and nerve fibres, especially on the peripheral elements, which in this case are the more susceptible (Holden 184, 185). The vascular change is secondary and not the real cause of the visual loss. This view is supported by a comparison between the

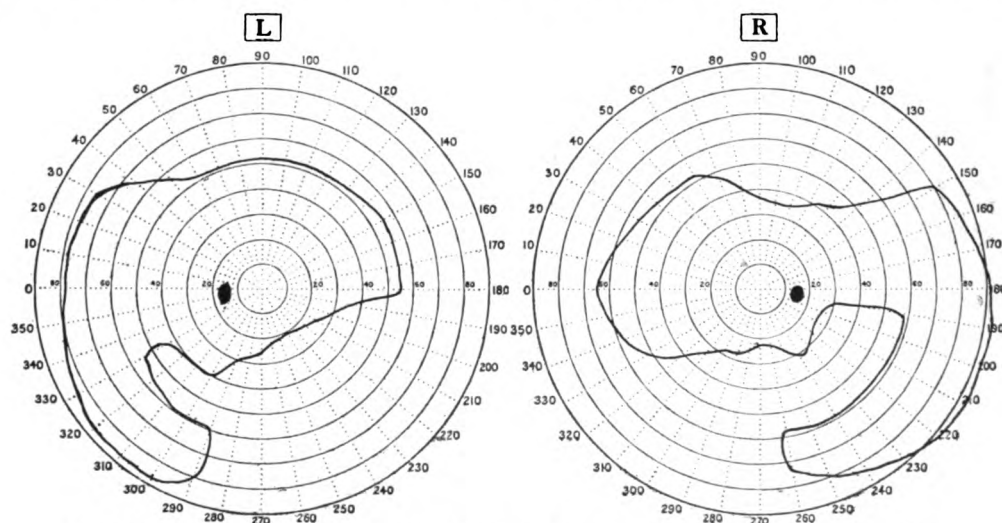


FIG. 100.—QUININE AMBLYOPIA. SAME CASE AS FIG. 99. FOUR WEEKS AFTER POISONING.

Note peculiar symmetrical defects. Object $\frac{3}{35}$. A 60-mm. object made the fields no larger, and a 1-mm. very little smaller. Colour vision proportionately affected so that not much more recovery is to be expected.

contracted vessels and the field changes, which does not show that the vascular changes are absent from those parts of the retina in which vision is retained. Moreover, the vision fails before the contraction appears and recovers while the contraction is progressing, while in a few cases the vessels are said to have remained normal in appearance. The presence of a central scotoma indicates a search for possible complication.

Diagnosis depends, as a rule, more upon the history and the ophthalmoscopic appearances than upon the fields, although the peripheral depression differentiates this form of toxic amblyopia from the scotomatous type. Special susceptibility to quinine is a prominent factor and a history of a small dose does not contraindicate the diagnosis. Somewhat similar field defects may be found in individual cases of tabetic atrophy or hysteria, or in the more rare condition of peripheral interstitial retrobulbar neuritis, but the history and mode of onset offer differentiating features. In hysterical amblyopia the fields may show a similar steep-edged contraction with good central vision, but the absence of orientation difficulty and the normal ophthalmoscopic appearances easily decide the diagnosis. In fresh cases difficulty can hardly arise unless for some reason

the facts are concealed by the patient ; in old cases in which the question of causation is raised in connection with compensation, pensions, etc., the determination of the field changes is of great value.

Prognosis.—Some restoration of sight may always be expected and the ultimate state of the vision is usually better where the early stages have not been very severe, and especially when the stage of complete blindness, if present, has not lasted long. The presence of disproportion indicates that further recovery may be expected, and even after several months some improvement may still take place. Apparent complete recovery of peripheral as well as of central vision may occur with remaining pallor of the discs, but the true state of the fields must be carefully tested by the screen before deciding that the recovery is really complete. Permanent total blindness has not been recorded.

Optochin or ethyl-hydro-cuprein, a derivative of quinine, offers an interesting example of the result of an alteration in the chemical constitution of the toxin. While idiosyncrasy is important here also, optochin is considerably more toxic to the visual neurones than quinine. The field changes run a course similar to those of quinine poisoning, but are distinguished by the relatively frequent occurrence of central scotoma with or without peripheral changes, a feature of extreme rarity in connection with the former drug.

Salicylic acid and salicylates have occasionally produced a condition resembling quinine poisoning. The field changes are similar but not so intense, the duration is shorter and the prognosis quite good.

The field changes due to *Filix mas* are not well known in detail. The whole field is affected and when the fields can be taken concentric contraction is found (Uththoff). The condition resembles quinine amblyopia, but the prognosis as regards permanent blindness is worse.

Another interesting example of the result of a chemical change is afforded by the organic preparations of arsenic. Whereas the inorganic pentavalent preparations rarely produce field changes, which consist of central scotomata when they do occur, some organic trivalent compounds such as soamin, atoxyl and others, manifest a high toxicity for the visual nerve elements and produce peripheral contraction resembling that caused by quinine, central scotoma being exceptional. In a review of thirty-seven cases of soamin poisoning in twelve of which details of the fields are given, Igersheimer found restriction of the field on the nasal side "almost without exception," and in no case a central scotoma at any stage. Moreover, this form of amblyopia is distinguished by the absence of the tendency to recovery, the cases practically all progress to complete blindness once the process has commenced, and the prognosis is therefore thoroughly bad. On the other hand, acetylarsan (Strobanti et Schepens, 391) and tryparsamide may produce field changes closely resembling those of quinine, with preservation of central vision and loss of orientation, the fields being almost tubular, together with contraction of the retinal arteries, and which may be followed by a degree of recovery similar to that which occurs in quinine poisoning.

C.P.

x

B. RETROBULBAR NEURITIS

Under this heading will be considered the field changes in disease of the optic nerve of inflammatory origin.

Three chief types have been recognised by Wilbrand and Saenger, axial neuritis, perineuritis, and total transverse neuritis, characterised respectively by dominant perimetric features, central scotoma, peripheral contraction, and more or less uniform and severe failure over the whole field. This classification, though not entirely satisfactory, is convenient and useful for the time being.

Acute Retrobulbar Neuritis or Acute Axial Neuritis

The common form of acute retrobulbar neuritis clinically familiar to ophthalmic surgeons is characterised by sudden or rapid failure of central vision in one eye, rarely in both, with, in many cases, few or even no concomitant symptoms. The examination of the field of vision is therefore an important factor in the study of this disease. Females are rather more commonly affected than males; one-half of the patients are less than forty years old and nearly all less than fifty. The lesion takes the form of a plaque, similar to the plaque of multiple sclerosis, which is itself the most usual cause, situated in the optic nerve and affecting the papillo-macular bundle. If it is near the eyeball swelling of the optic disc may be present, in which case enlargement of the blind spot will be added to the defects caused by the conduction interference with the nerve fibres. Often, however, the fundus oculi is normal in appearance. The incidence of the lesion as an initial manifestation becomes less and less frequent farther up the visual path; it occurs occasionally at the junction of the nerve with the chiasma, sometimes in the chiasma, very rarely in the tract, and apparently never at a higher level. The lesion develops rapidly and then more slowly resolves leaving the axis cylinders still capable of conduction so that vision may be restored. Its site and course determine the position and behaviour of the field defects and the main clinical features are therefore unilateral incidence, rapid onset and gradual recovery.

The chief and most characteristic field change is a defect in the central area. This scotoma is usually fairly large, some 10° to 20° in diameter, but may be more extensive so that a comparatively narrow zone of more or less unaffected field remains peripherally. A small scotoma of a few degrees only may occur in slight cases, but is much less common than a pronounced defect, and, when found, indicates the necessity for careful examination and watching in order to establish a correct diagnosis. Small central scotomata of this nature occurring at the commencement of the process are not commonly met with in practice, partly on account of the rapidity of development, and partly because advice is rarely sought before the defect has become comparatively gross.

In position the scotoma is central, though by no means always pericentral. The main part of the defect is often paracentral, the fixation area being eccentrically placed within it, but usually well included, and the centrocæcal type is common.

The shape is often approximately circular, but may be oval or irregular. Sector defects in the form of vertical or horizontal hemianopic or quadrant changes may be present, and sometimes the scotoma is cut off sharply along the vertical meridian of the

field. An important example of this type is the quadrant scotoma, occupying the apex of one quadrant, frequently the upper temporal, which may be termed "junction" scotoma (Figs. 107, 108) as it indicates that the site of interference is at the junction of the optic nerve and the chiasma. Arcuate defects extending from the blind spot may form incomplete or entire ring scotomata and indicate the involvement of fibres in the nerve which in the retina form bundles arching around the macula. Ring scotomata of zonular type may be large and dense with a very small relatively preserved central field at the fixation point, like an island of vision in the blind area. Smaller ring scotomata unconnected with the blind spot occur occasionally, but are rare except during recovery.

The intensity of the scotoma varies considerably. As a rule, the visual loss is severe or even absolute, and if the defect is also extensive, such cases can only be examined by the confrontation method or with very large test-objects. Less frequently the use of colours or of very small white objects may be required to demonstrate the defect, and here the same considerations apply as in the case of very small scotomata. Any intermediate degree may be met with. The degree of blindness in the area of the defect is not uniform. There are usually one or more areas or nuclei at or near its centre in which the loss of vision is most severe. In contra-distinction to this common feature the zonular ring scotoma (Figs. 119, 120) with relative preservation of central vision affords an interesting and hitherto unexplained contrast. In other cases analysis with serial test-objects may split up the defect into quadrants thus indicating the site of the causal lesion or may disclose other special features of diagnostic value.

The margins of the scotoma are usually steep, more rarely sloping and the gradient may vary at different parts.

The peripheral field may be unaffected, but will not infrequently be found to show an indentation of the isopters towards the place where the scotoma extends farthest out, and the latter often breaks through to the periphery by a *col*, or neck, which may easily escape observation unless several isopters are marked out. In severe cases there may be large and obvious gaps affecting the outer parts of the field.

The field changes rapidly attain their maximum extent and intensity, and for this reason observations upon the early stages are rarely made. A strong tendency to spontaneous recovery is characteristic. During recovery the defects gradually shrink and disappear, vision for white returning more rapidly than that for colour. As improvement proceeds the character of the defect may alter and features previously obscure, owing to the intensity of the visual failure over the whole field, may become manifest. A completely blind field may resolve by peripheral recovery into a central scotoma with peripheral depression. The rate of recovery varies considerably, good vision is frequently restored in two or three weeks, but sometimes the defect may persist for months before ultimately disappearing. At first recovery is often gradual, and in some cases after some days of slow progress a sudden or rapid increase of central vision may occur. If the field be tested at this time a ring scotoma will often be found, as if central vision had risen up in the middle of the depressed area. There is for the most part no definite correlation between the condition of the optic disc and the state of the field. In many cases the disc is normal and gross changes are rare. Later on a certain degree of pallor

develops and remains permanent although central vision may have recovered and the field may appear normal. In such cases careful examination will nevertheless usually show some depression of vision.

The bilateral forms differ in several ways from the unilateral type. This group includes Leber's disease, neuromyelitis optica, retrobulbar neuritis in meningo-encephalitis, and other conditions. Swelling of the optic disc is more frequent and may simulate plero-cephalic oedema so that cases have been recorded in which a cranial decompression operation has been undertaken. The eyes are affected together or one a short time after the other. The disease begins more gradually than the unilateral type, though in some cases bilateral blindness may develop very rapidly, and is more severe and persistent and does not exhibit in such a high degree the tendency to recovery. Most

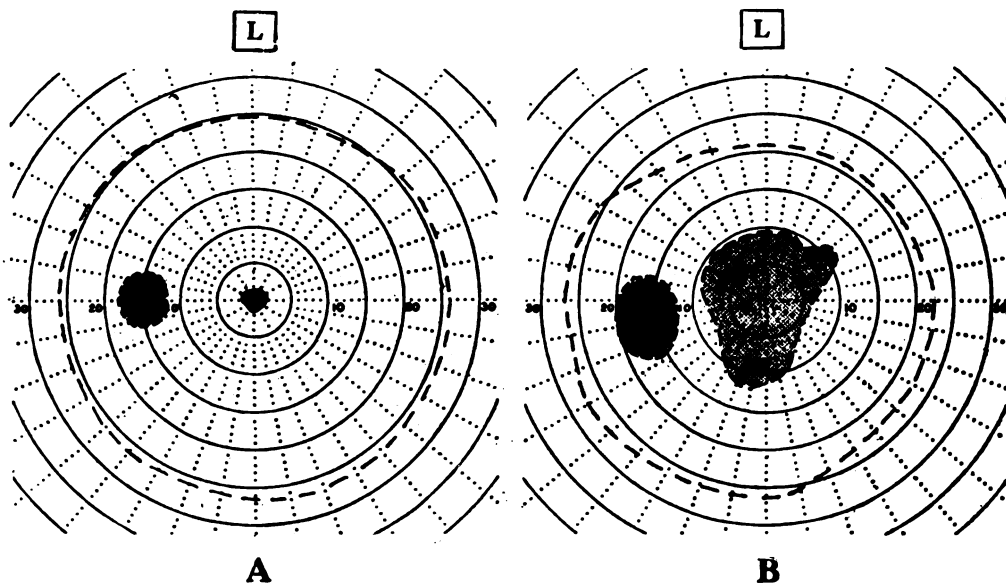


FIG. 101.—RETROBULBAR NEURITIS.

- A. Normal central field with minute central scotoma for $20/60$ white, slightly larger for $20/60$ red. V. 60 .
 B. Central scotoma for $20/60$ white. Peripheral isopters for $20/60$, $33/60$ and $33/60$ normal. V. = 60 nearly. (M. 1913.)

cases retain some permanent defect of sight and the prognosis is much worse than in the unilateral type. The chief clinical features in this group are bilateral incidence, moderately rapid onset, slow progress and tendency to permanent visual defect. The field changes are similar to those of the unilateral type but usually of a grosser character. The scotomata may be large, with irregular and sometimes severe involvement of the peripheral field. Nerve fibre bundle defects are rare and indications of chiasmal or tract interference may occur.

Chronic Retrobulbar Neuritis

Apart from chronic forms of toxic amblyopia such as that due to tobacco which are probably retinal, chronic retrobulbar neuritis is a comparatively rare disease. Some forms of retrobulbar neuritis run a slow clinical course; such cases are more frequently bilateral than the acute type, and although some recovery often occurs, optic atrophy

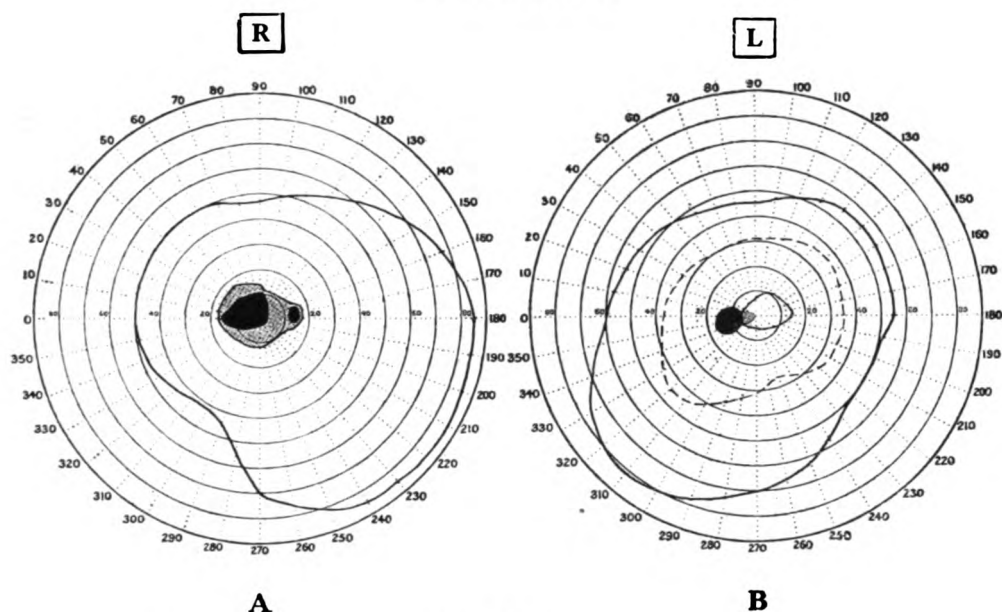


FIG. 102.

- A. Common form of scotoma in retrobulbar neuritis, including blind spot and a dense nucleus. Objects $\frac{3}{2} \frac{5}{0} \frac{0}{0}$ and $\frac{6}{2} \frac{0}{0} \frac{0}{0}$. V. = Fingers at 1 m. (D'A., 1923.)
 B. Depression of field shown by contracted isopters for $\frac{5}{3} \frac{5}{0}$ and $\frac{3}{3} \frac{3}{0}$. Central field for $\frac{5}{1} \frac{5}{0} \frac{0}{0}$ green shows defect merging into a centrocaecal scotoma. V. $\frac{5}{1} \frac{5}{0}$. (P., 1911.)

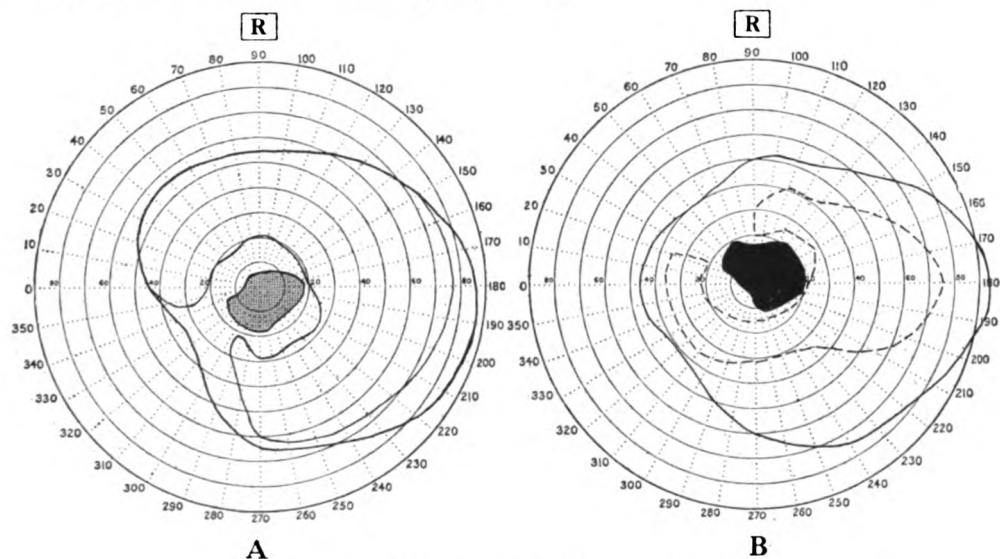


FIG. 103.—SCOTOMA WITH BREAKING THROUGH.

- Objects : A. $\frac{1}{3} \frac{0}{3} \frac{0}{0}$ peripheral field and scotoma, $\frac{5}{3} \frac{5}{0}$ inner field. V. = Hand movements. (W., 1914.)
 B. $\frac{3}{3} \frac{3}{0}$ scotoma and peripheral field, $\frac{3}{3} \frac{3}{0}$ broken line. V. = Fingers at 1 m., eccentric. (B. B., 1913.)

with ultimate serious visual defect may result. They are liable to be confused with visual impairment due to pressure on the optic nerve and with tobacco amblyopia. The field changes resemble in a general way those of the acute form.

Interpretation.—The characters of the field defects correspond approximately with

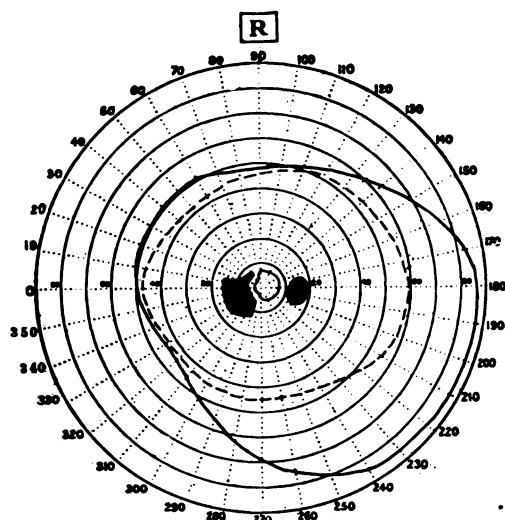


FIG. 104.—RARE TYPE OF FIELD CHANGE IN RETROBULBAR NEURITIS.

Central field depressed; scotoma with small nucleus to nasal side of fixation area. Blind spot enlarged owing to swollen disc. Vision never below $\frac{3}{8}$ part. Objects: periphery $\frac{3}{8}$, $\frac{3}{8}$; central field $\frac{3}{8}$. Blind spot $\frac{3}{8}$. Scotoma $\frac{3}{8}$. (H., 1925.)

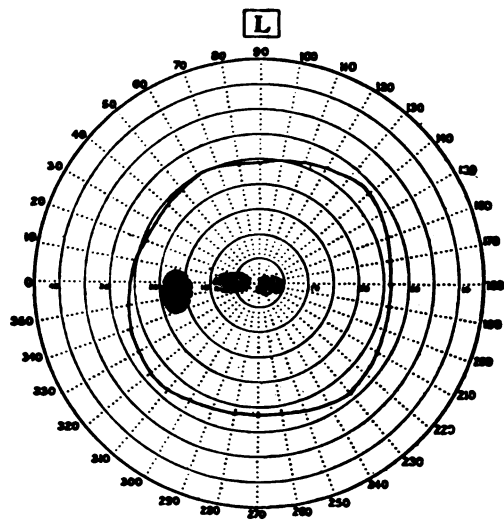


FIG. 105.—CENTROCAECAL SCOTOMA IN STAGE OF RECOVERY.

The scotoma has become divided freeing the fixation area. Objects: $\frac{3}{8}$, $\frac{3}{8}$, $\frac{3}{8}$. V. = $\frac{3}{8}$ +. (A., 1925.)

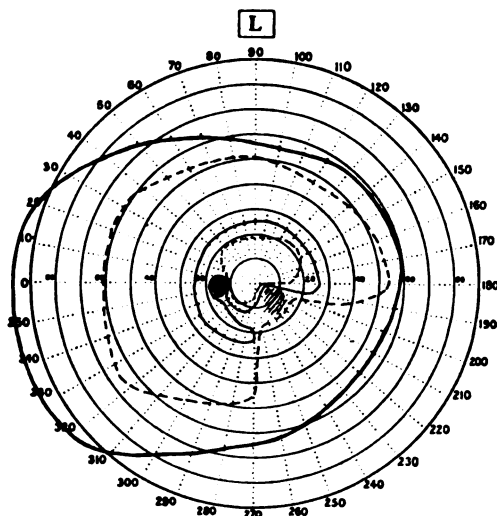


FIG. 106.—NERVE FIBRE BUNDLE DEFECT IN UNILATERAL RETROBULBAR NEURITIS.

Lower nasal quadrantic defect passing up to blind spot. Defect for red extends nearly to fixation point. V. $\frac{3}{8}$. Objects: $\frac{3}{8}$, $\frac{3}{8}$, $\frac{3}{8}$, $\frac{3}{8}$, $\frac{3}{8}$. (H., 1934.)

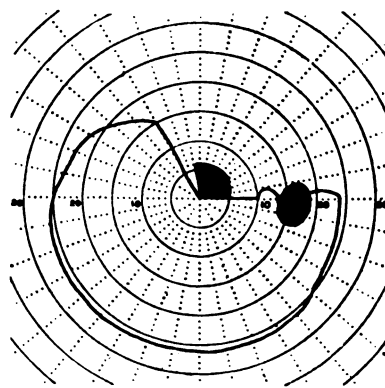


FIG. 107.—JUNCTION SCOTOMA.

Field $\frac{3}{8}$, scotoma $\frac{3}{8}$. V. $\frac{3}{8}$ part, with rapid fluctuations. Left field normal. Young man, cause unknown. (S., 1919.)

the position, shape, and extent of the lesion and the degree of interference which it causes. The centrocaecal position of the scotoma may be due to a lesion situated either in the retina or in the nerve. According to Wilbrand and Saenger a centrocaecal scotoma breaking through to the nasal periphery corresponds to a lesion in the anterior end of the

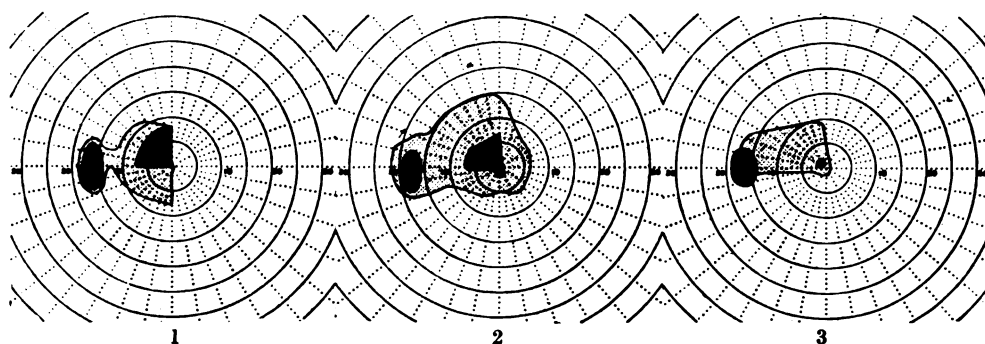


FIG. 108.—JUNCTION SCOTOMA, SHOWING VARIATIONS ON (1) MAY 25TH, 1914, V. = $\frac{1}{16}$; (2) JUNE 1ST, V. = FINGERS AT 2 M.; (3) JUNE 3RD, V. = $\frac{1}{4}$ PLUS.

Periphery normal. Field of right eye normal. Objects 1000 and 1000 . Young woman, cause unknown. (R., 1914.)

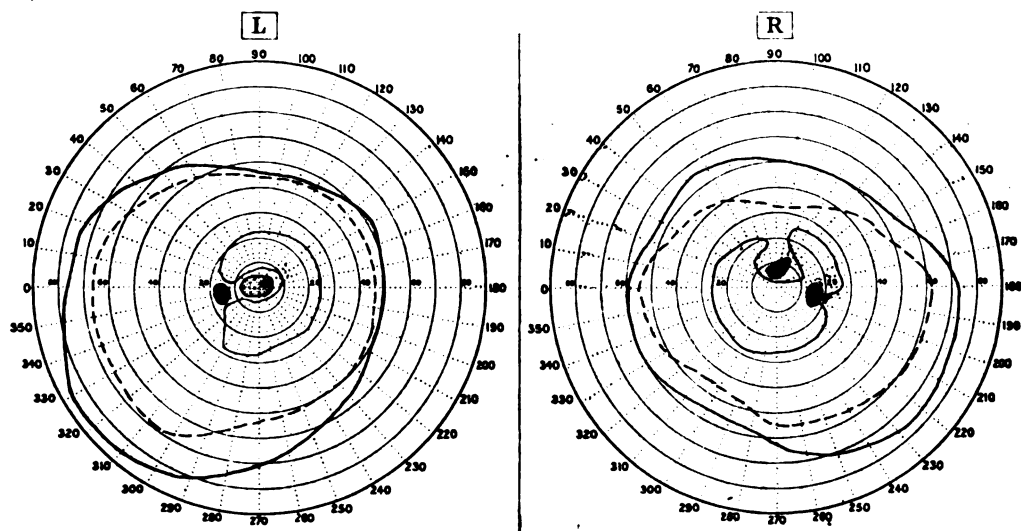


FIG. 109.—BILATERAL RETROBULBAR NEURITIS WITH ASYMMETRICAL DEFECTS, AND SOME PERIPHERAL DEPRESSION.

Defect centrocæcal in one field and supracentral in the other. Objects 330 and 330 , 2000 and 2000 . Male, age 41, cause unknown. R.V. $\frac{1}{16}$: L.V. $\frac{1}{16}$. (L., 1922.)

nerve where the papillo-macular bundle and the fibres from the lateral retinal periphery lie close together (see Fig. 30). The presence of nuclei in the scotoma and the varying incidence of the visual defect, central, paracentral or peripheral, suggest that special susceptibility is a factor in the distribution of the field changes. Anatomical conditions in connection with blood supply probably also have an important influence. The ring scotoma of zonular type which may be present throughout the course of a retrobulbar neuritis or only during the period of recovery is difficult to explain. It probably depends in some way upon the conditions of blood supply to the nerve fibres and the manner in which these are modified by the inflammatory focus, or it may be related to the great superiority of central over paracentral vision. There is no feature in the field changes which localises the lesion at any particular point between the anterior and posterior ends of the nerve, though its chiasmal termination is indicated by the junction scotoma. Nerve fibre bundle defects are rare: while they have no special significance

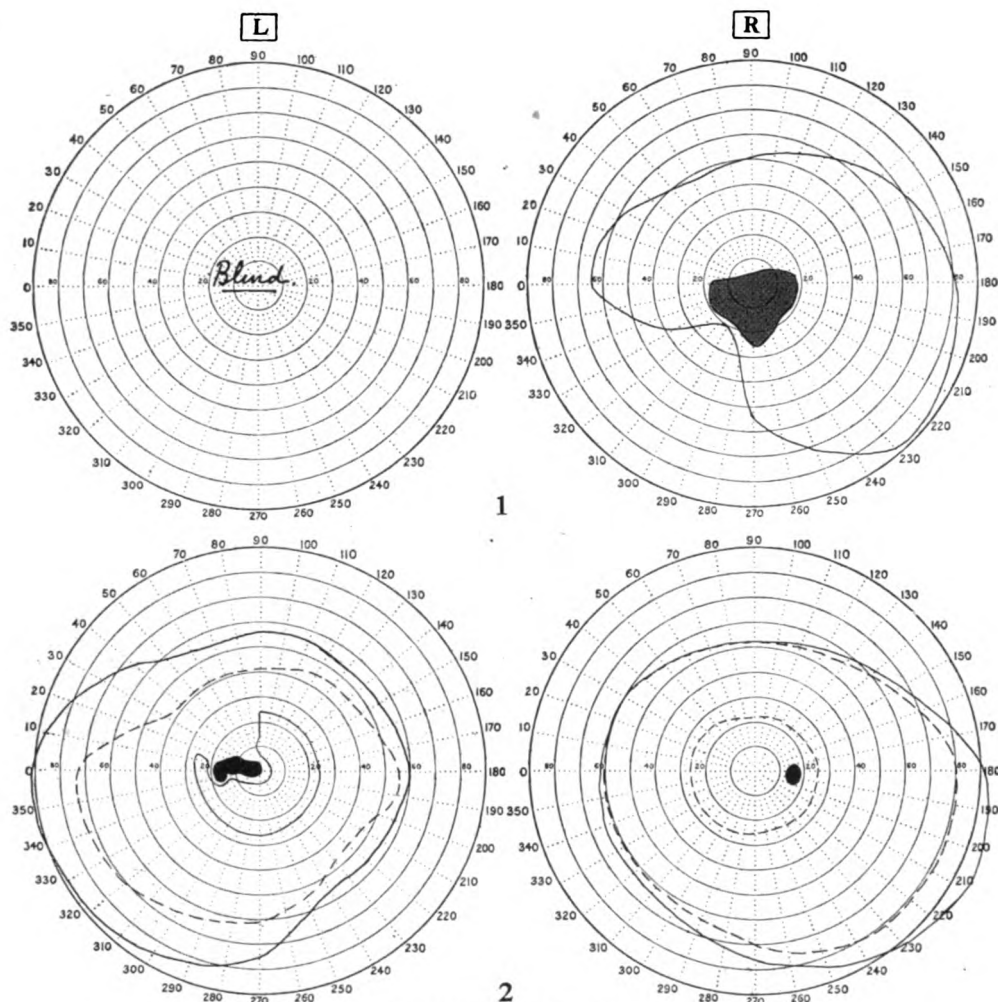


FIG. 110.—BILATERAL RETROBULBAR NEURITIS IN A CASE OF SYPHILIS.

1. Right field for $\frac{30}{30}$, showing large scotoma with peripheral sector defect. V. = Fingers at 1 ft. Left eye blind.
2. Six months later. R.V. = $\frac{6}{6}$ —. Field normal. L.V. = Fingers at 2 m., centrocaecal scotoma. (P., 1922.)

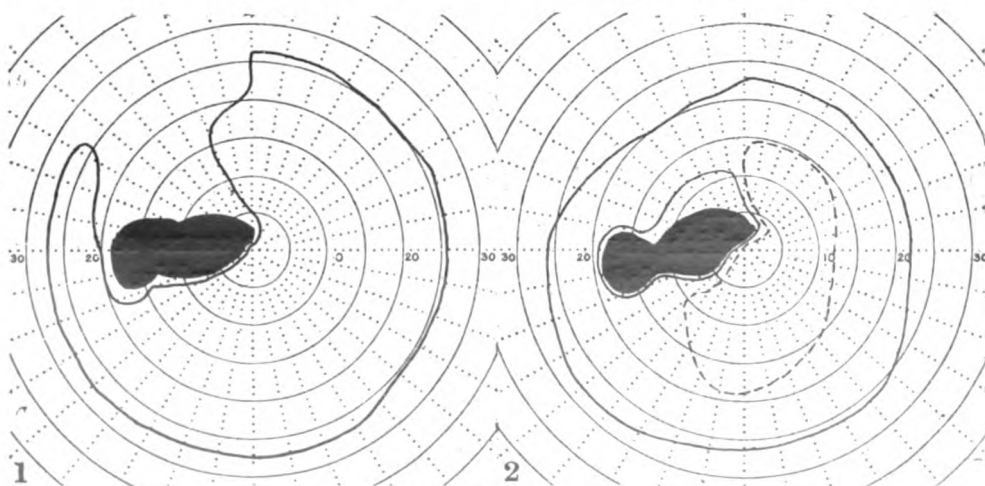


FIG. 111. RECOVERY. DETAIL OF SCOTOMA IN FIG. 110, 2, LEFT FIELD.

- (1) Six months later, V. = $\frac{6}{15}$ part; (2) two years later, V. = $\frac{6}{12}$. Objects $\frac{20}{200}$, $\frac{20}{200}$, and $\frac{20}{200}$. The fixation area has gradually cleared and the field for $\frac{1}{2000}$ has appeared.

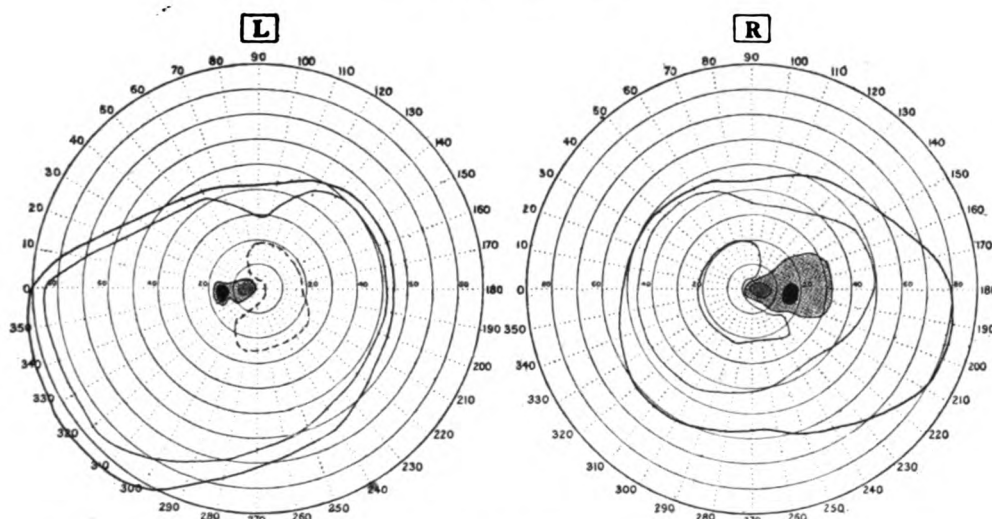


FIG. 112.—BILATERAL RETROBULBAR NEURITIS WITH SYMMETRICAL CENTROCÆCAL DEFECTS.

The scotoma resembles that of tobacco amblyopia in shape and position, but there is much more depression of the fields. Periphery for $\frac{30}{30}$ and $\frac{30}{30}$. Central field $\frac{20}{500}$. Scotoma, R.E., $\frac{20}{500}$, with nucleus for $\frac{30}{30}$; L.E. $\frac{30}{500}$, with nucleus for $\frac{20}{500}$. R.V. $\frac{30}{30}$; L.V. nearly $\frac{30}{30}$. Female, age 43, cause unknown. (M., 1914.)

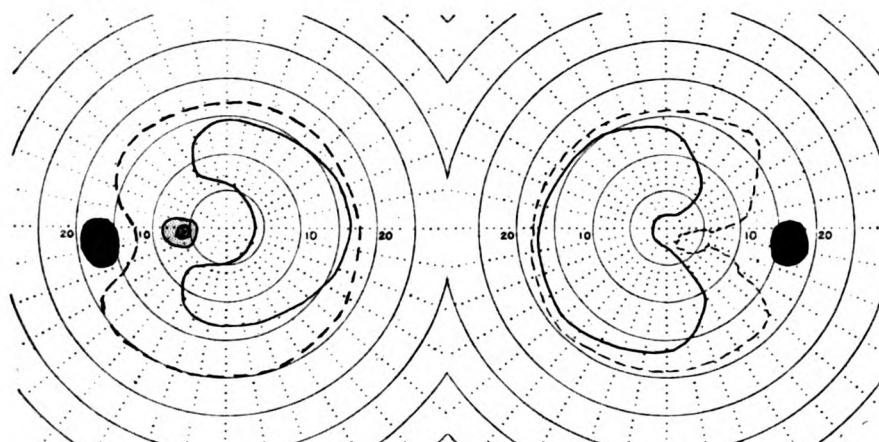


FIG. 113.—SAME CASE AS FIG. 112 SEVEN MONTHS LATER.

Central fields only: periphery normal. Vision R. and L. $\frac{30}{30}$. Central field for $\frac{20}{500}$ red. Peripheral field $\frac{20}{500}$ white contracted, with centrocæcal defects. Good vision with central colour defect.

for diagnosis or prognosis they show that fibres which lie together in the retina remain closely associated in the nerve. They are probably due to a specially localised conduction block depending on vascular interference. Disproportion indicates that the damage to the nerve elements is to a large extent only partial.

Diagnosis.—The principles of anatomical and pathological interpretation, which have already been discussed, are utilised to determine as far as possible the site and the nature of the causal lesion. While these problems can only be elucidated by the consideration of the complete clinical picture, the perimetric evidence is of great value, since in many cases the visual symptoms form the only or almost the only obvious deviation from normal conditions, and they may help to indicate the direction of investi-

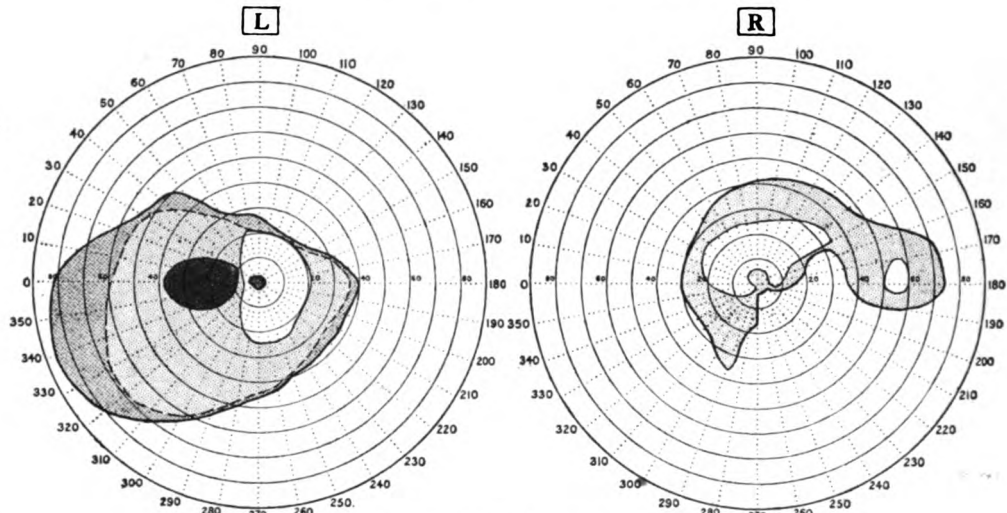


FIG. 114.—BILATERAL RETROBULBAR NEURITIS WITH IRREGULAR FIELDS.

Male, age 34, cause unknown. The charts show fields five years after onset; general nervous disease appeared five years later. V. = Fingers at 1.5 m. (C., 1913.)

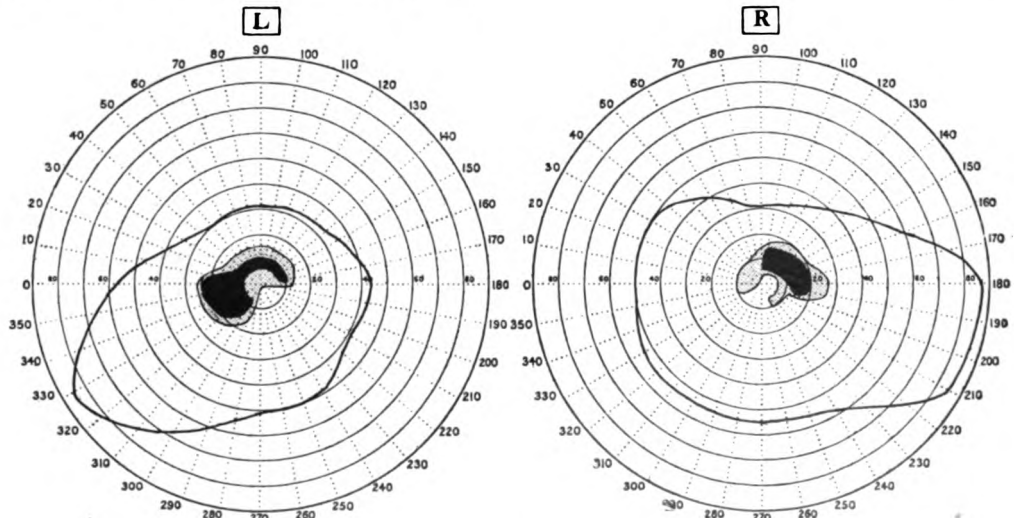


FIG. 115.—RETROBULBAR NEURITIS WITH BILATERAL NERVE FIBRE BUNDLE DEFECTS.

Objects $\frac{1}{3} \frac{1}{5} \frac{0}{0}$ and $\frac{1}{3} \frac{1}{5} \frac{0}{0}$. Male, age 21, cause unknown, possibly Leber's disease. R.V. $\frac{0}{6} \frac{0}{0}$. L.V. $\frac{0}{3} \frac{0}{0}$. (R., 1912.)

gation. In order to obtain the information which perimetry can offer the examination should not stop, as it so often does, at the mere detection of the presence and size of the defect, but should include full investigation of both fields and analysis of the changes found. The ophthalmoscopic examination should be very careful, and any changes found should be correlated with the visual symptoms, bearing in mind that a normal disc does not exclude retrolbulbar neuritis nor does a swollen disc necessarily indicate its presence. When the scotoma is very small and relative macular disease must be carefully excluded.

Diagnosis in retrolbulbar neuritis is essentially the problem of the explanation of a

scotomatous field defect not associated with retinal changes in the macular area. The first questions which arise are :—To which of the groups of optic nerve disease does the case belong and is it of unilateral or of bilateral type ? After thoroughly examining the characters of the scotoma the field of the other eye should be closely investigated especially if analysis of the scotoma has disclosed quadrantic or hemianopic features. A very slight and easily overlooked hemianopic defect may be present in the apparently unaffected field, or a scotoma of the ordinary central or paracentral type. The discovery of such defects indicates that the case is one of chiasmal disease or one of the forms of bilateral retrobulbar neuritis.

Since defects of chiasmal origin are usually in a more advanced stage in one field than in the other, theoretically there may be a unilateral defect in the earliest stage. Only the subsequent involvement of the other field can differentiate such a lesion from one in the termination of the nerve, and it must also be remembered that a lesion may begin in the termination of the nerve and spread to the chiasma.

Wilbrand and Saenger point out that a lesion at the chiasmal termination of one nerve producing a field defect on that side, if near enough to the chiasma, may cause a peripheral defect in the upper temporal quadrant of the opposite field on account of the involvement of the “knee” of crossed fibres from the opposite nerve. Thus although one nerve only is concerned both fields are affected. If the lesion is sufficiently severe there may be complete blindness on the affected side and a small upper temporal defect on the opposite side. The actual part played by the “knee” factor seems, however, somewhat doubtful.

Should the other field prove to be normal the lesion must be subchiasmal and confined to one nerve, provided only that we are not dealing with a case of the bilateral type which is affecting one eye earlier than the other. If the scotoma shows hemianopic or quadrantic features—junction scotoma—the lesion is probably at the chiasmal termination of the nerve. More commonly these features are absent and the defect is the ordinary central or centrocæcal scotoma representing the familiar form of unilateral retrobulbar neuritis.

When the field changes are bilateral their character indicates in the same way as in unilateral cases whether the site of interference is in each nerve or at a higher level. In such cases the detection or exclusion of hemianopic signs is usually easy. In bilateral retrobulbar neuritis the defects are frequently of similar extent and intensity in the two fields, although they need not be simultaneous, whereas in chiasmal interference a great disparity may be present. The distinction between the unilateral and bilateral forms is of great importance since the causes, clinical course, and prognosis are usually very different.

The diagnosis of the cause and nature of the lesion is often difficult and uncertain. Three factors are of material assistance :—The position and shape of the scotoma, the unilateral or bilateral incidence and the course and behaviour of the field changes. The importance of the clinical picture as a whole need hardly be stressed. The determination of the site is of value in relation to scotomata of the junction type for, as Velter has shown, the chiasmal termination of the nerve is a favourite site of multiple sclerosis. It is also

a part of the nerve which is exposed to local pressure, and the diagnosis usually lies between these two conditions, of which the latter is less common. The slower onset, persistence, and gradual increase of the pressure defect are also in strong contrast to the more lively behaviour of the defect due to inflammation. Below the termination of the nerve the same considerations apply, though, of course, not without exception. A somewhat hemianopic type of defect owing to the incidence of the interference at one side of the nerve is suggestive of pressure. In such cases there may be some peripheral field change in addition to the central defect (Fig. 136).

The usual irregularly rounded or centrocæcal scotoma, the ring scotoma and the nerve fibre bundle defect have no special diagnostic significance.

The behaviour of the field changes reflects the course of the lesion and in this way indicates its nature. The rapid development and slower resolution of the defect with pronounced tendency to recovery suggests multiple sclerosis—by far the most common cause—or a solitary nerve lesion of similar type. Field defects of similar character due to septic foci such as the nasal sinuses or the teeth, or to vascular disease, are relatively uncommon and tend to be more persistent, especially when the cause is pressure on the nerve.

If the field changes are unilateral the whole group of bilateral optic nerve affections is excluded, excepting always those cases in which one nerve is attacked some considerable time before the other.

A definite and clearly demonstrable central scotoma, whether unilateral or bilateral, excludes hysteria.

When the field changes are bilateral, hemianopic features indicate a chiasmal, or in rare cases a suprachiasmal, lesion. If such features are absent one of the group of bilateral scotomatous optic nerve diseases is present. This group includes the toxic amblyopias so far as they are not retinal, Leber's disease, neuromyelitis optica and some related though obscure cases, atypical cases of multiple sclerosis, also a small number of cases associated with general disease or deficiency, such as syphilis, diabetes or avitaminosis, or following an infection such as influenza or typhoid. As already pointed out in some cases the scotoma appears in one eye first and only after a considerable interval in the other, or may remain unilateral. In differential diagnosis the severe type of tobacco amblyopia must be borne in mind, though it rarely presents difficulty. Bilateral retrobulbar neuritis due to a local cause such as nasal sinusitis is uncommon. Arachnoiditis is also a possible, though rare, cause.

Prognosis and treatment depend upon the discovery of the cause where this is possible, though in the ordinary acute unilateral case, in which so often no cause can be ascertained, the prognosis is good as regards recovery of sight. Even severe cases of this nature with gross field defects usually recover with little or no resulting visual loss, although there may be pallor of the disc. There remains, however, a definite liability to the subsequent development of nervous disease, particularly multiple sclerosis. A slow course with delayed recovery increases the probability of a serious permanent defect, especially if after a period of improvement a check occurs.

When the condition is bilateral the outlook is, as a rule, less hopeful, though in acute cases practically complete recovery may result. In chronic or slowly progressing bilateral cases the prognosis is usually serious, and worse the longer improvement is delayed. The possibility of serious disease of the nervous system as a sequel is also present.

Progress should be estimated by repeated field examinations rather than by testing central vision only. The relations between the fields for white and for colour should be noted, the presence of disproportion during recovery indicates that further improvement is to be expected. When apparent complete recovery has ensued the actual state of vision can only be ascertained by a field examination with small visual angles, although for practical purposes the estimation of central acuity by Snellen's test is usually sufficient.

Peripheral Interstitial Neuritis

This is a relatively rare condition of which I have had no personal experience, and the following description is derived from recorded cases. The field changes consist of peripheral depression and contraction with relative retention of central vision, corresponding to the inward spread of the disease from the pial sheath along the connective tissue septa. The advancing peripheral loss of vision with the absence of central scotoma produces a resemblance in perimetric features to the optic atrophy of tabes or to the secondary atrophy which follows plerocephalic oedema of the nerve.

The peripheral contraction is sometimes unilateral, but more commonly affects both fields, usually unequally. The visual failure begins gradually, and in the earlier stages the loss for white may not be marked unless for small visual angles, while the defect for colour is evident. As central vision is relatively well preserved, the patients often do not come under observation until the field changes are well advanced and the optic discs pale. In the later stages central vision fails also, and only a very small field may remain, which in some cases finally disappears, leaving complete blindness.

Sometimes the field is irregularly involved and sector-like defects varying in size and shape may be produced.

The site of the interference is usually in the posterior part of the nerve where the macular fibres lie centrally. A peripheral lesion situated where the central fibres are superficial, as in the anterior part of the nerve, might, on anatomical grounds, be expected to produce a nasal sector defect extending to the centrocæcal area, a relatively rare perimetric finding in retrobulbar neuritis. As the lesion may extend a little way along the nerve different sets of fibres may be affected at different points, and, beyond indicating its subchiasmal position, the field changes do not exactly locate the site of interference.

The peripheral form of retrobulbar neuritis occurs usually in connection with a perineuritis which itself may arise from several morbid conditions, either as a local process confined to the nerve, or as a descending neuritis in connection with meningitis or with arachnoiditis. If no other cause is obvious, syphilis should be suspected. In some cases the diagnosis between tabetic atrophy and syphilitic perineuritis is very difficult. In the former pallor of the optic discs accompanies, whereas, in the latter, it follows the visual failure. Very contracted fields approximately equal in size are more characteristic of tabetic atrophy than of peripheral neuritis in which the extreme contraction is often

unilateral. A decided degree of recovery excludes tabes (Wilbrand and Saenger). No special perimetric features are known to be definitely associated with the various causes and the differential diagnosis must be made on other grounds.

The prognosis depends upon the cause, the degree of optic atrophy present, and the extent and duration of the field changes. It is, on the whole, unfavourable, though good results have been obtained in syphilitic cases when treated at a sufficiently early stage. Owing to the relative preservation of central vision the severity and progress of the disease and its response to treatment are better gauged by perimetry than by sight testing in the ordinary way. The presence of proportion or disproportion between the fields for colour and for white gives useful information.

Total Transverse Neuritis

Wilbrand and Saenger's third group includes cases characterised by involvement of the whole cross-section of the nerve, giving rise to rapid amblyopia or amaurosis affecting the whole field. There is evidently no definite separation as regards the field changes between these cases and very severe examples of the first group. Sometimes if the visual loss is not too intense, or when a certain amount of recovery has taken place, a large and dense central scotoma may be demonstrated or careful examination with very large objects may show that the amblyopia is more intense centrally, indicating that the field changes are essentially of the same nature as those of the usual type of retrobulbar neuritis. The cases are mostly bilateral and may occur in connection with gummatous infiltration of the nerves, following an infection such as influenza, as an initial stage of neuromyelitis optica, or without discoverable cause. In cases of pronounced general depression without demonstrable central scotoma the tendency to recovery is less prominent and the prognosis less favourable than in the commoner scotomatous type.

Retrobulbar neuritis may be regarded as a secondary manifestation, the primary cause being either a local process in the neighbourhood of the nerve or a systemic disease. We may now briefly consider the field changes in retrobulbar neuritis associated with some of the more important of these conditions.

Multiple Sclerosis

In a series of 400 cases of retrobulbar neuritis Benedict suspected that 90 per cent. were due to multiple sclerosis.

The subgeniculate portion of visual nerve path may be affected by this disease at any level, and more than one focus may be present at the same time. Lesions are common in the optic nerves, less frequent in the chiasma, and rare in the tracts, and are important since they may occur at an early stage in the disease. Suprageniculate lesions occur only when the disease is very advanced.

The field changes are of the toxic-inflammatory type and occur in two forms, firstly those which are present in patients who have other manifestations of multiple sclerosis with moderate or slight defect of central vision, and secondly, those in which

severe loss of central vision is prominent with or without the presence of other signs at the same time.

Changes of the first variety have been found in the routine examination of the fields. They consist of multiple, small, relative scotomata, paracentral or central in position, characterised by indistinctness, slight intensity, and sloping edges, so that they are difficult to demarcate and best detected by small colour tests. Both fields are frequently affected, and there may be associated peripheral contraction. Such defects are described by Wilbrand and Saenger and Klingmann, and have also been observed by Paton and by Hensen.

A slowly progressive optic atrophy may be present in some cases with gradually increasing depression and peripheral contraction of the fields.

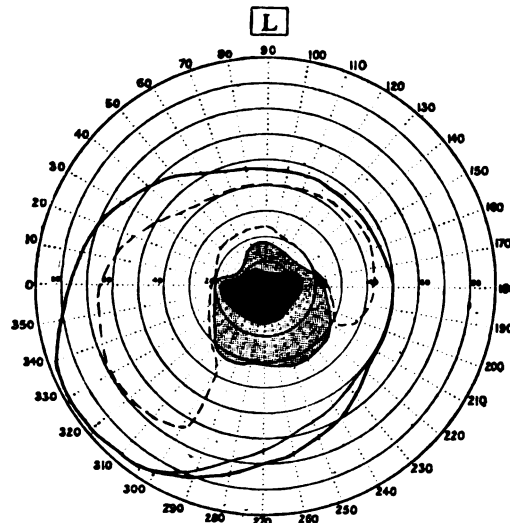


FIG. 116.—MULTIPLE SCLEROSIS. CENTRAL SCOTOMA WITH BREAKING THROUGH. Peripheral depression. Objects $33^{\circ}0'$, $33^{\circ}5'$ and $34^{\circ}0'$. Female, age 17. V = Hand movements. (P., 1913.)

More familiar to the ophthalmic surgeon is the second form in which the field changes are those of acute retrobulbar neuritis, the other symptoms being slight or absent at the time. The scotoma affects the field of one eye only and in a day or two attains its greatest extent and intensity. It is central or centrocæcal in position and irregularly round or oval in shape. The centre is usually absolute or nearly so and the intensity diminishes towards the periphery where the margins are moderately steep. Not infrequently the margins are very sloping at one part so that the central defect, especially if dense, may be connected with a peripheral depression by a neck or breaking through. Nerve fibre bundle defects have been observed by Roenne and ring scotoma by Uhthoff and Wilbrand and Saenger. The peripheral field may be unaffected or may show sector-shaped or irregular defects of varying intensity, so that the use of small visual angles is essential to determine its integrity.

Complete or nearly complete blindness of one eye may occur or, in severe cases, of the two eyes in sequence. Simultaneous blindness of both eyes is exceptional in multiple sclerosis.

When the site of interference is in the termination of the nerve, a unilateral hemianopic or quadrant scotoma may be produced, and, if higher up, hemianopic defects, bitemporal or homonymous, occur in accordance with the position of the lesion.

Junction scotoma is very suggestive of this disease.

As an uncommon variety, peripheral loss, usually somewhat irregular, occurs, the central field remaining relatively intact. Regular concentric contraction is rare and should be regarded with some scepticism as a direct manifestation of pure multiple sclerosis.

A characteristic feature is the variable and fluctuating course of the defects which may alter in position, shape, extent, or intensity, sometimes wandering from one part of the field to another or leaving one field to appear in the other, or recurring after disappearance. The tendency to recovery is very pronounced, and in a few weeks central vision by Snellen's test may be restored to normal, though a careful field examination may show slight defects still remaining. Hensen has observed that the scotoma nearly always recovers within three months if no other nerve symptoms are present or appear before the three months are completed. If other symptoms already exist or arise the scotoma tends to persist.

The behaviour of the field changes is in accordance with the course of the morbid process. The initial dense scotoma occurs in the stage of congestion and swelling; restitution of vision, with slight depression of the field or faint scotomata, corresponds to the stage of resolution with functional recovery of most of the axones, which are very resistant to this form of injury. The variability of the defect is due to resolution in one part of the lesion followed by the development of fresh foci elsewhere.

The diagnosis of multiple sclerosis, as far as the perimetric evidence is concerned, is suggested by the short duration and the inconstancy of the defects, or the presence of changes which seem to indicate more than one site of interference. Scotomata of the junction variety or showing other hemianopic features are also suggestive but require to be distinguished from field changes of the pressure type. Simultaneous or nearly simultaneous blindness of both eyes suggests one of the forms of bilateral retrobulbar neuritis such as neuromyelitis optica. Peripheral field changes, especially concentric contraction, with retained central vision, should be carefully differentiated from functional visual disturbances which may be found in cases in which symptoms suggestive of multiple sclerosis are present.

When the field changes pursue a more gradual and steady course they have, *per se*, no special positive significance in the clinical picture of multiple sclerosis, and the diagnosis depends upon other evidence. Multiple sclerosis is a very common cause of retrobulbar neuritis, of which probably 50 per cent. or more may be regarded as of this nature.

The prognosis as regards recovery of vision is extremely good, especially if the defect is of the acute scotomatous type, and the other signs of the disease are slight. Even very gross and intense defects rarely cause serious permanent visual loss. In the rarer gradually developing cases the outlook for recovery of vision is less hopeful.

Disease of the Nasal Accessory Sinuses

Although nasal sinus disease is an uncommon cause of retrobulbar neuritis, it is important in so far as its timely recognition enables correct treatment to be undertaken. The optic nerves are more liable to be affected by disease of the posterior than of the anterior sinuses, and the resulting neuritis is acute or subacute, seldom chronic, and is usually unilateral.

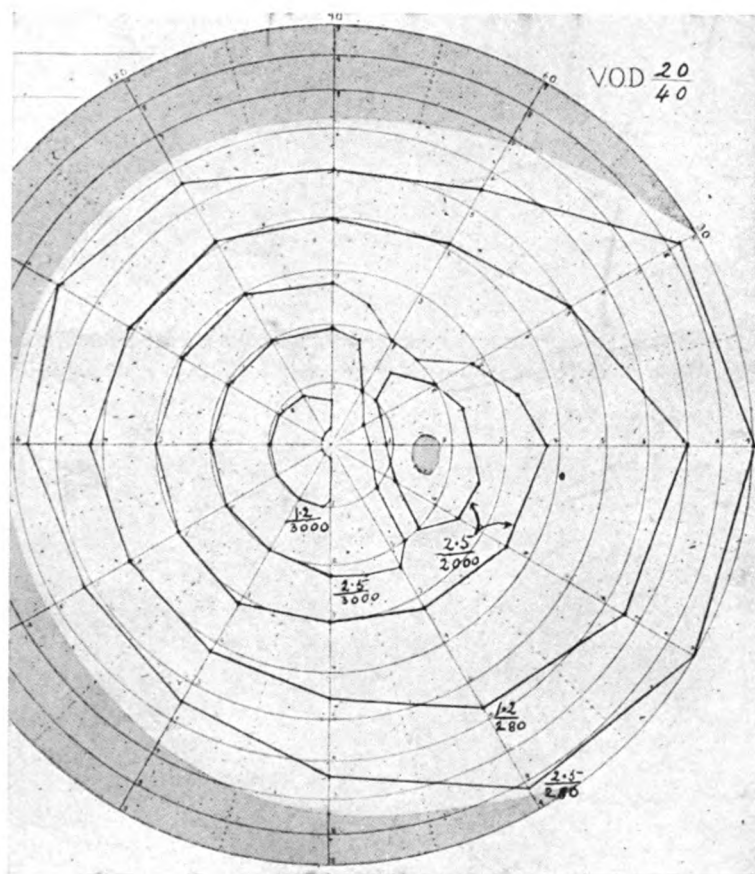


FIG. 117.—NASAL SINUS DISEASE.

Early changes. Pericæcal scotoma for $\frac{2.5}{3000}$. Temporo-central defect for $\frac{1.2}{3000}$. Vision $\frac{20}{40}$. (After Walker (452).)

Numerous observations have been recorded as to the character of the field changes present in this condition. These reported changes fall into three groups:—

1. Chronic cases of unexplained headache or visual impairment in whom latent sinus disease is suspected as a possible cause.

2. Cases of definite purulent inflammation of the sinuses without visual symptoms.

In these two groups three chief field conditions have been recorded: peripheral contraction, "enlargement of the blind spot" with normal field periphery, and normal fields. The field changes are not complained of by the patient, but found on examination.

3. Cases in which pronounced visual impairment is present. Sinus disease is usually manifest.

C.P.

N

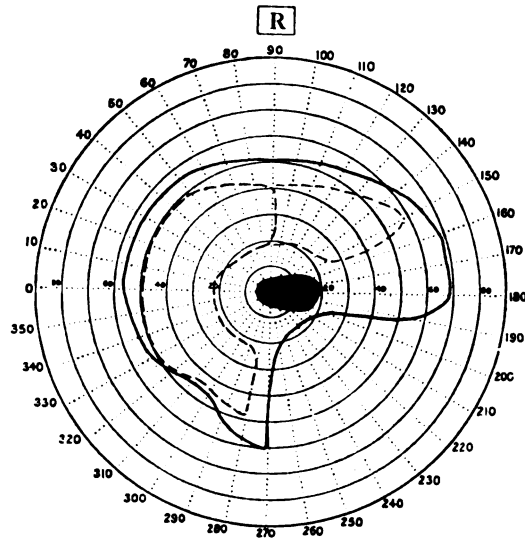


FIG. 118.—NASAL SINUS DISEASE. CENTROCÆCAL SCOTOMA WITH SECTOR DEFECT.

Objects $3\frac{3}{4}$ and $3\frac{1}{4}$. Vision = Fingers at 1.5 metres. After operation. Field could not be taken previously. (S., 1923.)

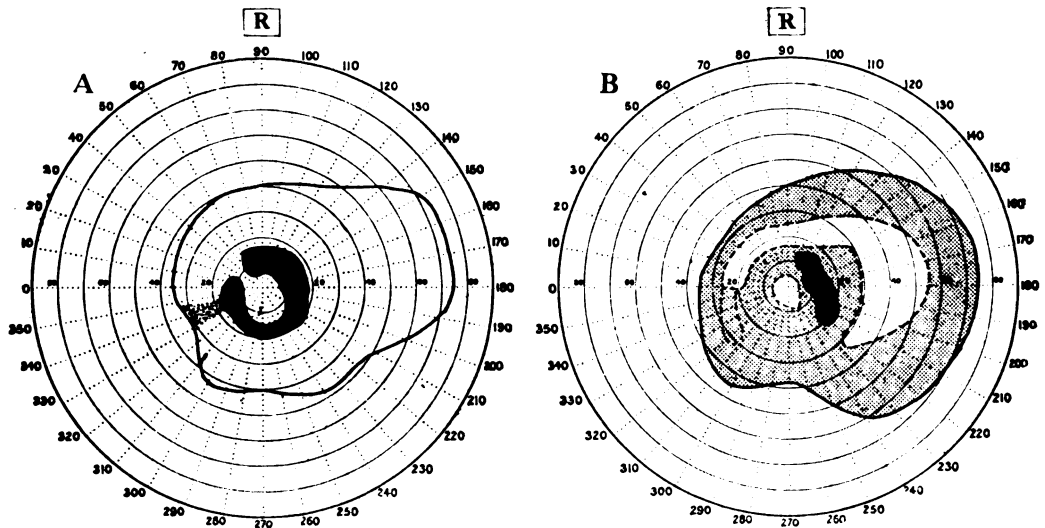


FIG. 119.—RIGHT POSTERIOR ETHMOIDAL ABSCESS. RING SCOTOMA. CONDITION BEGAN WITH ALMOST COMPLETE BLINDNESS OF RIGHT EYE.

A. Field taken three months after onset of symptoms and a few days after a preliminary nasal operation. Almost complete ring scotoma for $3\frac{3}{4}$. V. $2\frac{1}{4}$.

B. A week after opening of abscess. Appearance of central field for $3\frac{3}{4}$. V. $2\frac{1}{4}$ +. (H., 1923.)

Here definite changes of the toxic-inflammatory type, occasionally of the pressure type, are present; scotoma of the central field involving the fixation area to a greater or less degree with or without peripheral depression which, when present, is often local or irregular rather than concentric. The scotoma may also be of the centrocæcal or annular type. The defect may be progressive, leading to atrophy, or may fluctuate; it is persistent and does not follow the typical course of the scotoma in ordinary acute

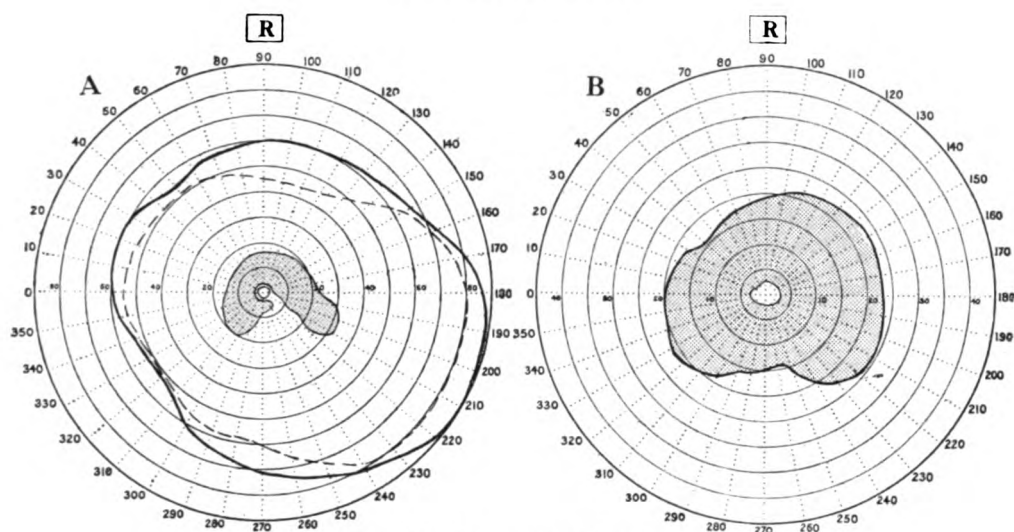


FIG. 120.—NASAL SINUS DISEASE.

A. Ring scotoma with gap below, $\frac{30}{0} \frac{0}{0}$. Periphery for $\frac{30}{0} \frac{0}{0}$, $\frac{30}{0} \frac{0}{0}$. V. $\frac{6}{8}$.
 B. Three days later: gap absent except for $\frac{20}{0} \frac{0}{0}$. V. $\frac{6}{8} +$. Chart on larger scale.
 Almost complete recovery one week after operation (V. $\frac{6}{8}$). (H., 1915.)

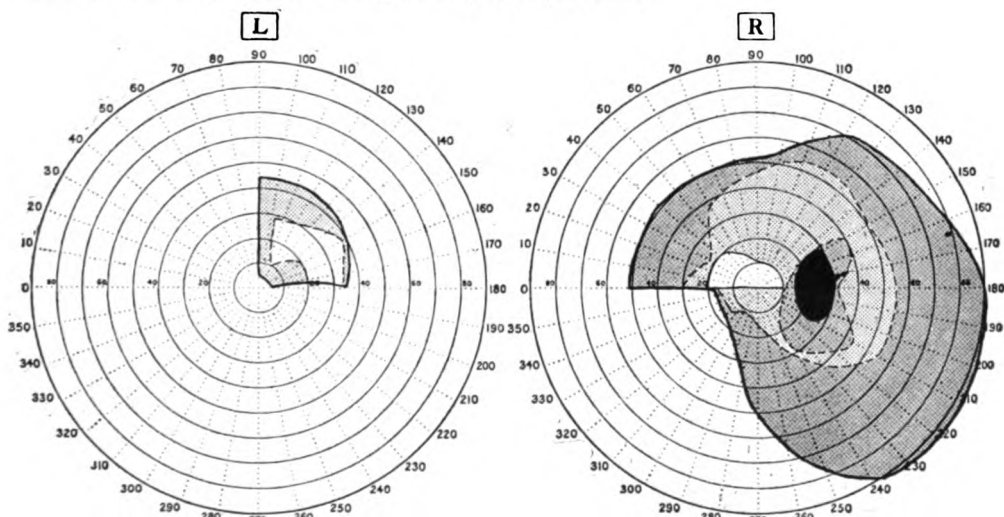


FIG. 121. SYPHILITIC DISEASE OF NASAL BONES.

Probably also gummatous disease affecting intracranial parts of the nerves and chiasma.

Left field retains only upper nasal quadrant; right field shows nasal step.

Objects: R. $\frac{40}{0} \frac{0}{0}$, $\frac{30}{0} \frac{0}{0}$, $\frac{30}{0} \frac{0}{0}$. V. $\frac{6}{8}$.

L. $\frac{30}{0} \frac{0}{0}$, $\frac{30}{0} \frac{0}{0}$. V. Hand movements. (E. K., 1915.)

retrobulbar neuritis. As long as the cause is active there is little or no tendency to recovery.

Interpretation.—A study of the literature in relation to changes in the first two groups indicates such a complete lack of agreement in the observations of different workers, and at the same time such a definite consistency in the findings of individuals that the conclusion is unavoidable that these results have not been uninfluenced by the method of examination and by the attitude of the observer. In the few cases of this

group which I have personally examined, I have found the boundaries and slope of the fields always within physiological limits of variation. If the bilateral field changes reported are common in sinus disease the apparently normal field in a case of unilateral retrobulbar neuritis of rhinogenic origin should usually show one or other of the alterations described. Such findings are not recorded, but I have personally observed field changes of obviously psychical nature in the field of the unaffected eye. Until the true nature of these field changes is more fully established, caution and reserve are necessary in drawing inferences from them as to the fibre architecture of the nerve or the way in which they are produced.

In the third group the changes indicate the presence of a retrobulbar neuritis, or, in rarer cases, of a pressure interference situated in the nerve. Although cases of bitemporal hemianopia have occasionally been attributed to sinus disease no real evidence exists to show that the crossed fibres are ever affected in the chiasma. Nor is it possible to say from the field changes whether the condition of the nerve is due to an actual extension of inflammation from the diseased sinus or to secondary results such as congestion or intoxication. The most acceptable hypothesis is that of venous congestion in the nerve, leading to impaired nutrition and suffocation of the nerve fibres.

Diagnosis.—In the majority of cases of rhinogenic retrobulbar neuritis the sinus disease is sufficiently evident to enable the diagnosis to be made. In the absence of demonstrable sinus disease a sceptical attitude should be adopted towards the possible rhinogenic origin of the field changes. When a rhinogenic neuritis is suspected as the cause of a scotomatous defect, the field changes can give little positive assistance as there is no special type of defect associated with sinus disease, but they are of considerable value in a negative sense, as by their study we are enabled to exclude many other causes, such as tobacco or hypophysial tumour for example, whose effects have often been confused with those of sinus disease, and in this way to narrow the area of the problem. In such obscure cases the diagnosis usually lies between “latent” sinus disease and multiple sclerosis or a pressure interference, and help will often be obtained from the observation of the behaviour of the field changes. The presence of hemianopic or quadrant defects is against inflammatory sinus disease. Concentric contraction, apart from conditions such as tabetic atrophy, is usually functional and cannot be regarded as evidence of a presumed toxic influence, emanating from a hypothetical latent or otherwise symptomless—except for headache—morbid sinus condition and acting directly upon the optic nerve. In cases of mucocele it may be possible to diagnose a pressure interference, but that the source of pressure is a dilated sinus is not indicated by the perimetric findings.

BILATERAL FORMS

Neuromyelitis Optica

An important and interesting form of bilateral retrobulbar neuritis is that associated with myelitis. Both sides are always affected, though one may suffer a few days, or, in rare cases, weeks before the other. Blindness may come on so rapidly that it is not

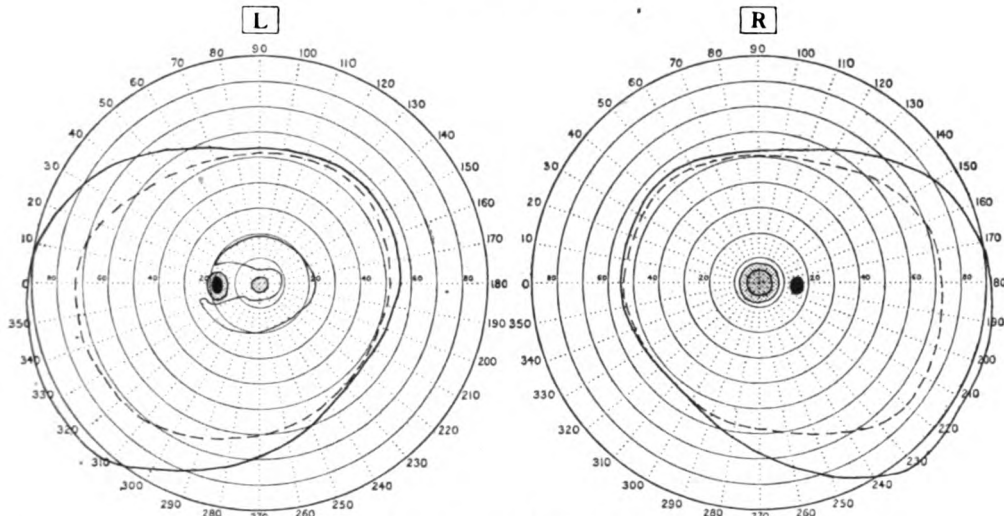


FIG. 122.—RETROBULBAR NEURITIS IN MYELITIS.

Female, age 13. Left field, centrocaecal scotoma: objects $\frac{3}{30}$ and $\frac{1}{30}$; $\frac{20}{200}$, $\frac{5}{200}$ and $\frac{2}{200}$. Right field, central scotoma: objects $\frac{3}{30}$ and $\frac{1}{30}$. (B., 1922.)

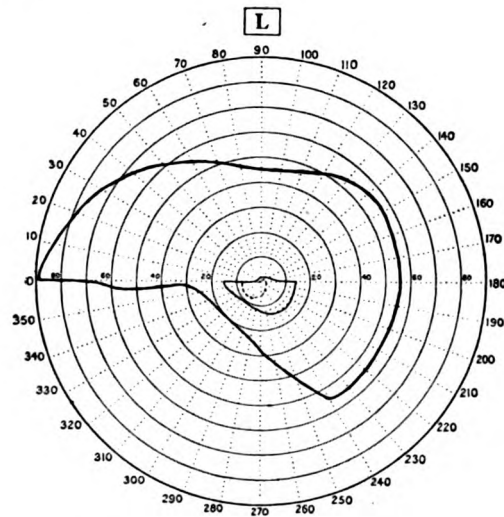


FIG. 123.—NEUROMYELITIS OPTICA.

Neuromyelitis optica in a girl of 13. Left field shows general depression with a large sector defect down and out and a central defect above the fixation point. V. $\frac{1}{15}$. Right field normal. V. $\frac{5}{5}$. A fortnight later both eyes blind.

Test objects $\frac{10}{30}$, $\frac{1}{30}$, $\frac{60}{60}$ red. (C. S., 1936.)

possible to investigate the fields, in other cases central or centrocaecal scotoma, quadrant or hemianopic central defects, sector defects, peripheral contraction or combinations of these have been reported. Homonymous hemianopia apparently does not occur. More rarely a moderate amblyopia of one form or another is present. In the cases with severe visual loss the character of the field changes may be observed during recovery as in a case observed personally in which, following complete blindness, central scotoma was present in one field and general depression, only slightly greater centrally, in the other.

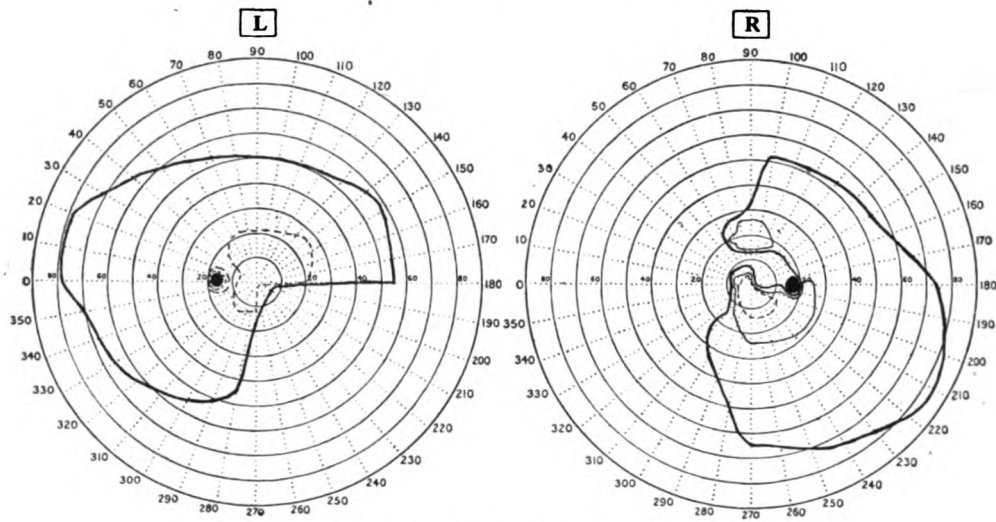


FIG. 124.—NEUROMYELITIS OPTICA.

Neuromyelitis optica in a patient 62 years of age. Loss of nasal field and arcuate scotoma R.E. Quadrant defect L.E. R.V. $\frac{6}{6}$. L.V. $\frac{6}{6}$. Objects R.E. $\frac{3}{30}$, $\frac{2}{200}$. L.E. $\frac{1}{30}$, $\frac{2}{200}$, $\frac{2}{200}$. (P.B., 1936.)

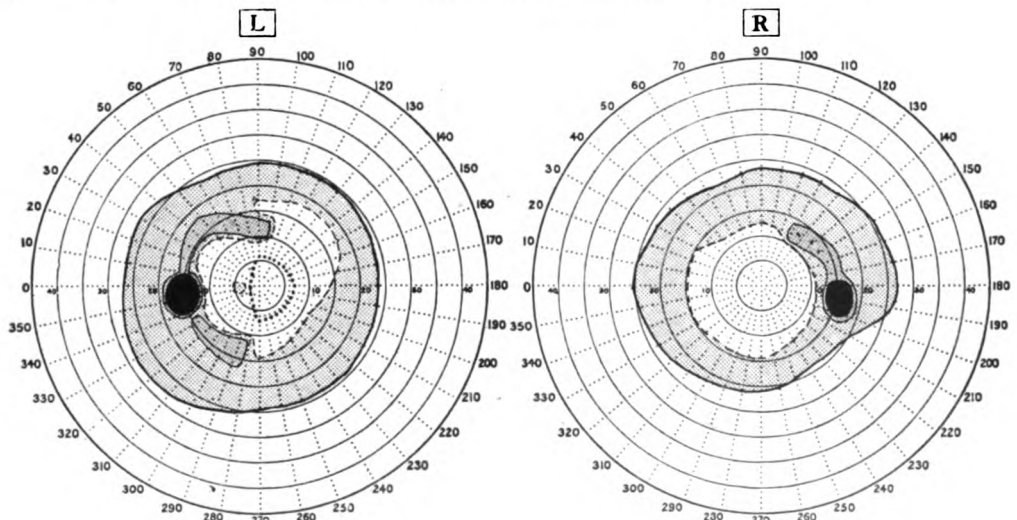


FIG. 125.—RECOVERY IN RETROBULAR NEURITIS, LEAVING DEPRESSION OF CENTRAL FIELD AND RELATIVE NERVE FIBRE BUNDLE SCOTOMA.

Field for $\frac{3}{30}$ normal. R.V. = $\frac{6}{6}$ part, L.V. = $\frac{6}{6}$. Objects $\frac{3}{30}$, $\frac{2}{200}$, $\frac{1}{200}$. Red $\frac{1}{200}$ (dotted line) L.E. only. Charts taken four years after acute attack when R.V. = P.L. L.V. = counting fingers 1.5 metres. Recovery began within two months. (W., 1925.)

The field changes are very variable in form, and may fluctuate in intensity; no special type occurs. At least partial recovery of sight may be expected, and persistent total blindness is an uncommon result. The patient may recover vision but die of the myelitis which usually appears a few days or weeks after the visual symptoms, more rarely at the same time or beforehand.

The *post-mortem* findings are in agreement with the field changes, lesions having been observed in the nerves, chiasma and tracts, but not in the latter alone (Uhthoff).

Owing to the frequently intense amblyopia, perimetry does not play an important part in the diagnosis, except possibly in cases which are seen early or in which the field changes develop sufficiently slowly. Until the appearance of the spinal symptoms, it is not possible to diagnose this form of neuritis by the ocular symptoms alone. The occurrence of rapid bilateral blindness with swollen discs and negative evidence in other directions should arouse suspicion. As regards prognosis the development of severe visual symptoms would appear to indicate, though not without exception, that the spinal lesion will also be severe. The mortality is stated to be about 50 per cent. (Goulden).

Bilateral retrobulbar neuritis with rapid complete blindness followed by recovery sometimes occurs in children. Possibly these and some other cases of acute bilateral retrobulbar neuritis belong to the same group, or are closely related to the myelitis cases, the spinal affection being absent.

A similar form of retrobulbar neuritis occurs in acute disseminated encephalitis.

Hereditary Optic Atrophy or Leber's Disease

Leber's disease is a form of acute or subacute retrobulbar neuritis which is usually hereditary, occurring typically in the males of affected families about the time of puberty

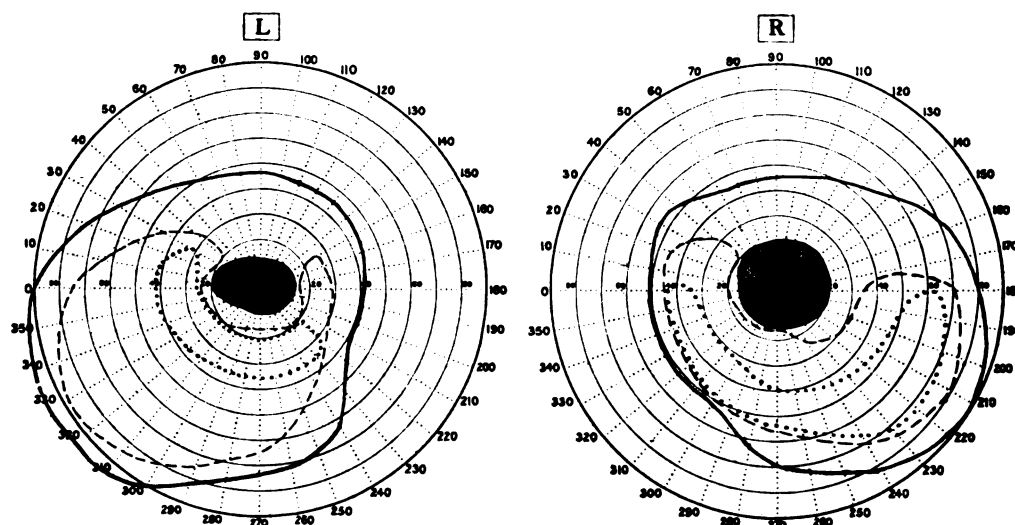


FIG. 126.—LEBER'S DISEASE. CENTRAL SCOTOMATA WITH SYMMETRICAL BREAKING THROUGH UPWARDS.

Objects: R. $\frac{3}{30}$, $\frac{3}{30}$, $\frac{3}{30}$; $\frac{3}{30}$ red. V. Fingers at 1 m.
L. $\frac{3}{30}$, $\frac{3}{30}$; $\frac{3}{30}$ red. V. Fingers at $\frac{1}{2}$ m. (H., 1923.)

or adolescence, or later at about forty to fifty years. Isolated cases without family history also occur, and females are occasionally affected, more commonly in the latter of the two age periods.

The failure of sight begins, as a rule, with moderate rapidity, and increases for a few months, after which gradual improvement occurs leaving usually a considerable defect, while complete blindness is exceptional. The tendency to recovery, so charac-

teristic of acute unilateral retrobulbar neuritis, is only slight. Both eyes are affected usually about the same time or, less frequently, with an interval of weeks or months.

Owing to the bad central vision fixation is uncertain and there is often difficulty in accurately mapping out the field defects. Every precaution to exclude error is required.

The characteristic feature is a central scotoma, varying in size but usually some 15° to 40° in diameter, round or oblong in shape, occasionally irregular. In position the defect is approximately pericentral and not commonly distinctly paracentral, *i.e.*, with the fixation area near its edge. The centrocaecal type of scotoma may be present, well overlapping the fixation area. Definite quadrantic or hemianopic features do not occur. The intensity varies, as a rule the defect is very dense or absolute in the most depressed part which is usually near the centre of the scotoma, while the surrounding

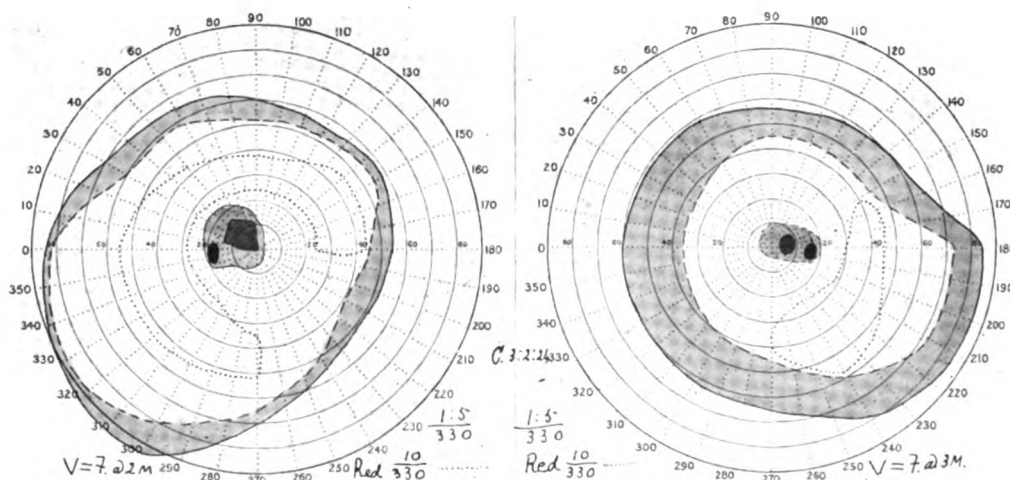


FIG. 127.—LEBER'S DISEASE; BREAKING THROUGH INWARDS FOR COLOUR ONLY.

area is less affected, so that the edge presents a fairly steep slope. The scotoma is multiform; many different forms and shapes may be found in a series of cases and it may be quite different in the two fields. Peripheral vision for colour and white is often impaired, though a moderately large white test-object may show a normal boundary, and frequently a sector-shaped or irregular depression connects the periphery with the scotoma.

This breaking through may occur at any part of the field, but is more common in an inward, or upward and inward, direction and may give rise to field conditions which resemble various forms of hemianopia. Should the peripheral depression appear symmetrically in the upper temporal quadrants a superficial resemblance to one of the stages of bitemporal hemianopia may result.

Nerve fibre bundle defects may be present, and in a case recorded by Hancock a small ring scotoma ultimately developed.

Interpretation.—The characters of the field defect, especially the absence of quadrant or hemianopic features, indicate that the site of interference is subchiasmal and the existence of nerve fibre bundle defects points to the nerve rather than the retina. Symmetrical breaking through of the scotoma into the upper temporal field quadrants should not be regarded as evidence of bitemporal hemianopia without verification. In behaviour the field changes correspond to the toxic inflammatory type ; no features suggestive of pressure are present.

Diagnosis.—The field changes place the condition in the bilateral group and when heredity can be established the diagnosis is made on that basis. In many cases no evidence of this kind can be obtained and the perimetric findings assume a greater importance. The important features are the rapid onset, bilateral field defects, slow progress and stationary final condition. On these grounds, and taking into consideration

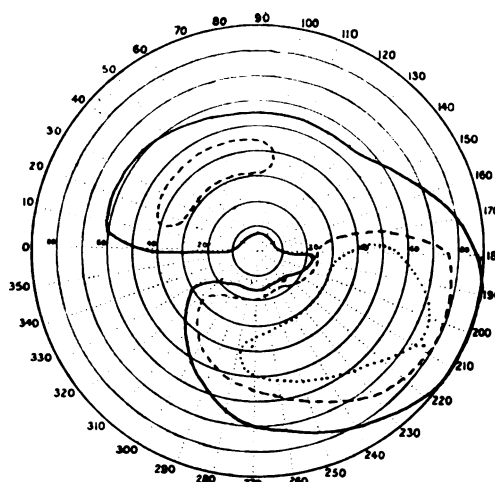


FIG. 128.—LEBER'S DISEASE, SHOWING NERVE FIBRE BUNDLE TYPE OF DEFECT. (FROM ROENNE (324).)

the general clinical picture, a case may be regarded as being of the Leber group. Months or years may elapse before the subsequent development of some other nervous disease necessitates an alteration of opinion.

Cases which present the general features of Leber's disease may be confused with tobacco amblyopia and with chiasmal interference but are usually easily differentiated by the dense steep edged scotoma with bad vision and without hemianopic features. The constant features of the tobacco scotoma contrast with the more multiform changes in Leber's disease. Local peripheral depression of the field, which does not occur in tobacco amblyopia, may be present.

Arachnoiditis in the optico-chiasmal region may also give rise to bilateral central scotomata and other field changes and should be borne in mind as a possible cause in cases in which the diagnosis is difficult.

The prognosis in cases of the Leber type, as well as in proved Leber's disease, is almost always unfavourable as regards perfect recovery of central vision, but is fairly good in respect of the possibility of serious blindness. *Ceteris paribus*, cases associated

with intense and extensive field changes both central and peripheral, and which run a protracted course, offer the least hopeful outlook.

The question of differential diagnosis between tobacco amblyopia, Leber's disease, and chiasmal interference as from a hypophysial tumour or meningioma sometimes arises. The more important perimetric features may be summarised as follows :—

(1) **Tobacco Amblyopia.** Scotoma diffuse and relative with central nucleus or nuclei, does not extend far to nasal side of fixation area, nerve fibre bundle defects absent, central colour field may show an extensive temporal defect, peripheral field unaffected, quadrantic signs absent.

(2) **Leber's Disease.** Scotoma dense, freely includes fixation area, nerve fibre bundle defects may be present, peripheral field usually affected, quadrantic signs absent.

(3) **Chiasmal Interference.** Scotoma dense, occupies the apex of a quadrant, nerve fibre bundle defects may be present, peripheral field affected, quadrantic features present in both central and peripheral field.

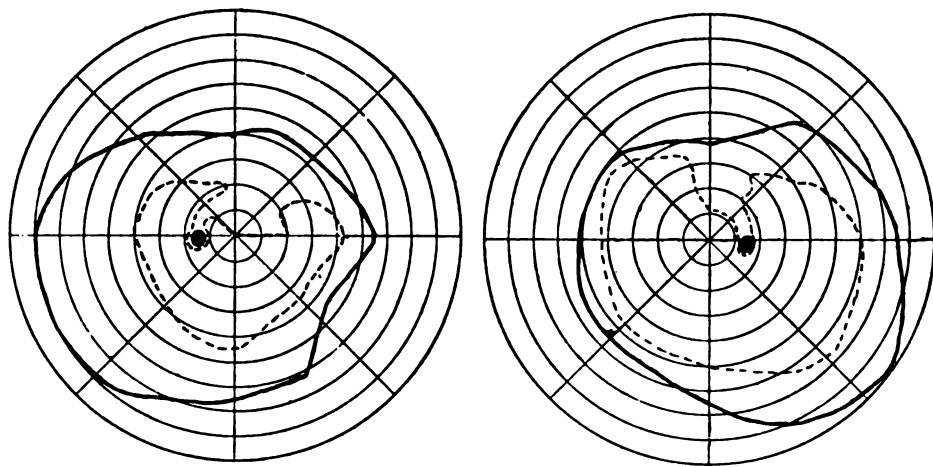


FIG. 129.—BILATERAL RETROBULBAR NEURITIS WITH NERVE FIBRE BUNDLE DEFECTS. EXACT NATURE UNASCERTAINED. FEMALE, AGE 43.

Field for $\frac{3}{30}$ nearly normal. Field for $\frac{3}{30}$ shows definite nerve fibre bundle defects. R.V. $\frac{6}{40}$. L.V. $\frac{6}{24}$. (G., 1923.)

Blindness following Hæmorrhage

This interesting condition appears to be a retrobulbar neuritis or an intoxication of the retinal ganglion cells (Holden 185). The patients are usually over forty years of age and have suffered from pre-existing disease so that their physical condition is below normal. Cases of war wounds with this ocular complication are, however, reported.

The usual cause is severe hæmorrhage from the gastro-intestinal tract or uterus. Within a day or two of the hæmorrhage rapid failure of vision occurs, bilateral in about 90 per cent. of the cases, and the optic discs are seen to be swollen, sometimes with small hæmorrhages.

Much depression of the field occurs with wide sector defects, which may produce

various forms of restriction resembling irregular hemianopia, vertical or horizontal, and always include the fixation area. Pincus believes that loss of the lower half of the field is so frequent as to be characteristic. Occasionally a central scotoma with intact

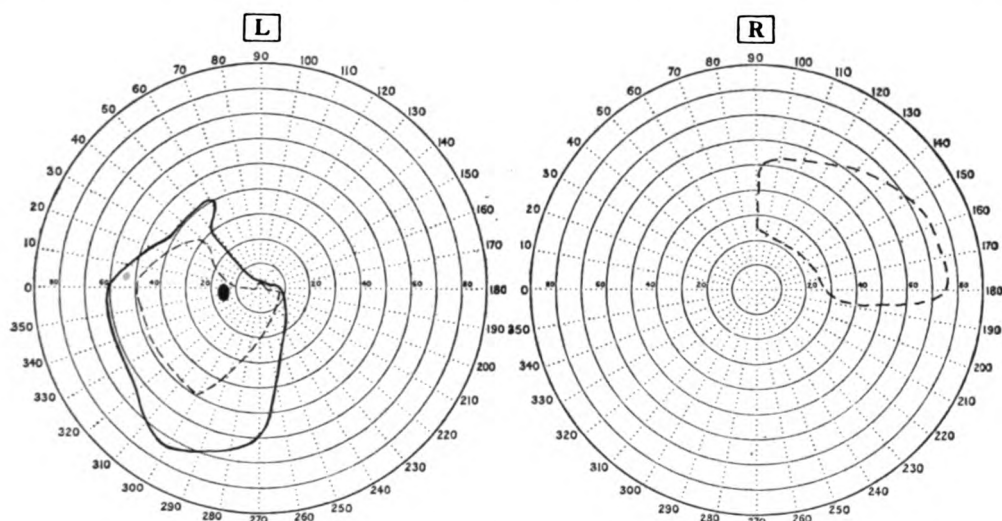


FIG. 130.—ATROPHY FOLLOWING GASTRIC HÆMORRHAGE. THREE MONTHS AFTER ONSET.

Vision in right field reduced to the perception of large white objects in upper temporal quadrant. Left field: objects $3\frac{3}{4}$, $3\frac{1}{4}$. V. $1\frac{1}{4}$. (M., 1924.)

peripheral field limits has been recorded. The changes may be very much more severe in one field than in the other and in the less affected field there may be depression of the central isopters. The defect is absolute or very intense, and although the tendency to recovery is present, the damage is so severe that a permanent loss of vision remains. Cases of complete recovery have, however, been reported.

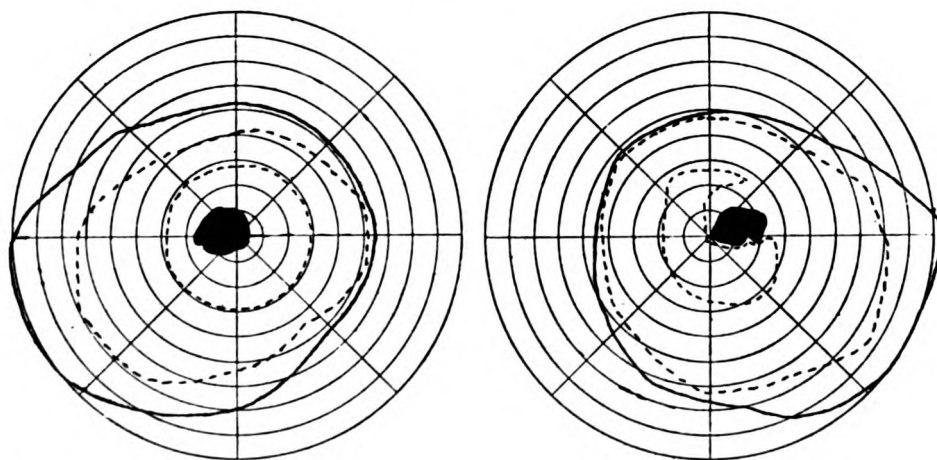


FIG. 131.—BILATERAL RETROBULBAR NEURITIS IN PERNICIOUS ANÆMIA WITH SUBACUTE DEGENERATION OF THE SPINAL CORD.

Fields for $3\frac{3}{4}$, $3\frac{1}{4}$, and $2\frac{3}{4}$. Scotoma $2\frac{1}{4}$. R.E. V. $\frac{2}{80}$. L.E. V. $\frac{1}{80}$. (G., 1932.)

Pernicious Anæmia with Subacute Degeneration of the Spinal Cord

In this condition bilateral retrobulbar neuritis may occur either before, about the same time as, or following the anæmia and cord symptoms. The visual defect may precede the anæmia—or the discovery of the anæmia—by some years. The field changes are similar to those of Leber's disease and do not present any distinguishing features. The onset is gradual and there is little or no spontaneous tendency to recovery. Complete blindness does not result, but permanent impairment of vision remains. The diagnosis depends upon the detection of the anæmia and cord symptoms. The prognosis is better when the anæmia is recognised early and suitable treatment instituted.

Diabetes

The scotomatous field changes produced by endogenic toxins in diabetes may be shortly referred to, as they are believed by many to resemble closely those of tobacco poisoning. Sometimes this is the case when the defect is centrocæcal in position, but in such cases the scotoma is denser and has steeper edges than that of tobacco, while the onset and course are different. The defect due to tobacco is constant in its characters whereas that due directly to the toxins of diabetes is multiform, and often pericentral and varying in shape. Any illness may cause the development of amblyopia in a smoker who consumes enough tobacco and diabetes is not infrequently a factor in the production of tobacco amblyopia.

We have seen that in tobacco amblyopia the central part of the field surrounding the scotoma undergoes certain changes. Changes of this constant and characteristic type are not found in diabetic amblyopia. Cases of pure diabetic toxic amblyopia have not hitherto been sufficiently closely examined in sufficient number to establish the true characters of the field changes, and in many of the reported cases the diabetic origin of the amblyopia is not convincingly demonstrated. Retrobulbar neuritis which can be ascribed to diabetes is extremely rare since the establishment of modern treatment for the disease.

Progressive Optic Atrophy, Tabetic Type**Tabes**

This form of atrophy is primary in the ophthalmoscopic sense, since the optic disc shows no signs of having passed through a stage of swelling and congestion. Without closely examining the vexed question of the pathogenesis of tabetic optic atrophy, we may note that it is probably due either to the action of the toxins of the spirochæta pallida directly upon the nerve fibres, or to an impairment of the nutrition of the fibres by a very chronic vascular and interstitial inflammatory process in the pial sheath and its trabecule, or to a combination of both these factors in varying degrees (Paton). That is to say, as far as we know, the factors concerned are essentially very much the same as those which act in other toxic inflammatory affections of the nerve.

Both nerves are always affected though in unequal degrees. The disparity varies greatly, it may be very slight or so pronounced as to suggest the integrity of the less affected field. In the initial stage it is probable that the condition is in some cases

unilateral for a time, but, as in other similar bilateral visual defects, the patient does not seek advice until the condition is more developed.

Following Uthoff (424) we recognise two chief types of field change :—

- I. General depression fairly equally distributed over the field.
- II. Local depression, the affected area being usually sharply limited from the remainder. The local depression may take various forms.

I.—It has been pointed out (Chapter V) that in general depression the internal isopters—those for colours and small visual angles for white—first show contraction, and this is evident in the early stage of the tabetic type of field change. Tested by the older methods, peripheral loss for colour was found with uncontracted fields for white. As the condition progresses the depression increases, and by the time that central vision has fallen so far as to incommode the patient the peripheral field will show relative contraction

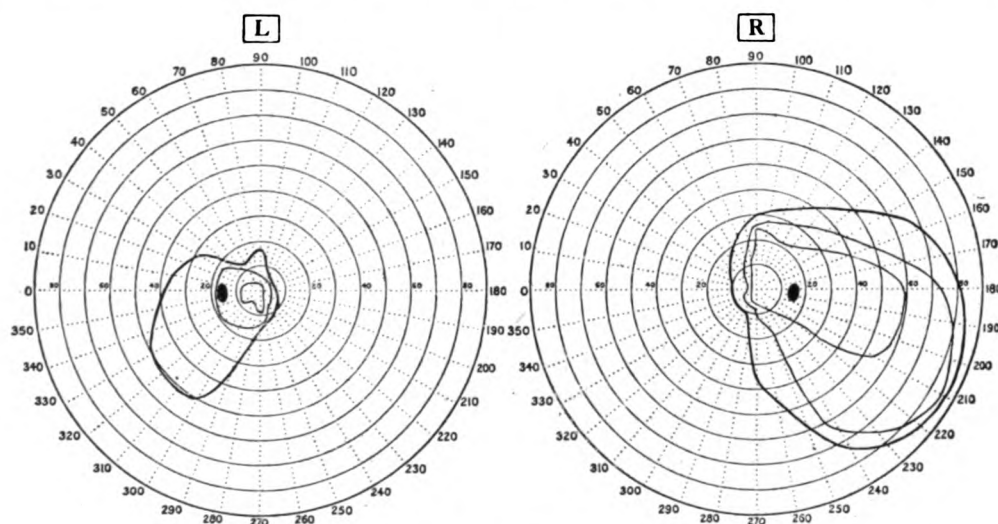


FIG. 132.—TABES.

Peripheral depression with relative retention of central fields. Symmetrical loss of nasal fields.

R.V. $\frac{6}{12}$. Objects : $\frac{50}{330}$, $\frac{20}{330}$, $\frac{4}{330}$.

L.V. $\frac{6}{12}$. Objects : $\frac{50}{330}$, $\frac{20}{330}$, $\frac{10}{2000}$. (G., 1922.)

for white, though often unrestricted limits if the object used is large enough. In rapidly progressive cases disproportion is pronounced, and colour vision, even at the centre, may be almost entirely lost except for large and brilliant objects, while central vision by Snellen's test remains good. Red-green blindness appears first, and is followed by blue-yellow blindness when the failure for white has become manifest.

II.—In the second type sector defects of various kinds occur with more or less steep edges whose slope may vary at different parts. The unaffected areas may retain good vision for a considerable period. Such defects may involve one or several quadrants and may produce a field picture resembling different varieties of hemianopia. A nasal step may be present, or an arcuate defect of nerve fibre bundle type passing to the blind spot (Fig. 134). A definite variety is characterised by an approach to true concentric contraction (coast-erosion type) with preservation of central vision, even for colour,

until the contraction has become extreme. These fields resemble those attributed to perineuritis.

Central scotoma has been observed occasionally, and is, according to Uhthoff, who found it in 2 per cent. of his cases, by far the rarest of tabetic field changes. There is nearly always associated restriction at some part of the periphery, and the scotoma progresses, breaks through, and expands until the whole field is lost. There may be a central scotoma in one field with peripheral depression or another type of change in the other. Hemianopia, both homonymous and bitemporal, has also been recorded, but is not regarded by Uhthoff as a pure tabetic manifestation.

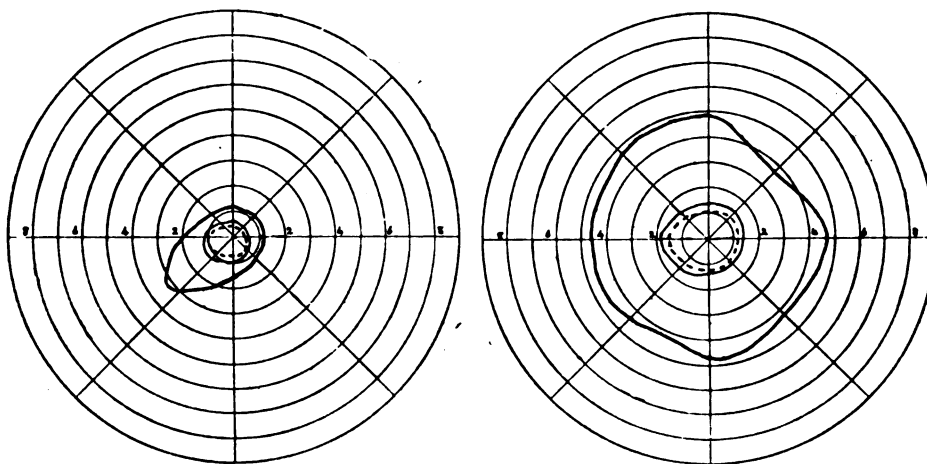


FIG. 133.—TABES.

Severe peripheral depression with retention of central fields illustrating the coast erosion type of concentric contraction. Each circle on the chart represents 1° .

R.E. Outer field for 2800 . Larger objects give no extension. Inner fields 2000 , 2000 red. V. $\frac{1}{2}$.

L.E. Outer field for 3680 . Larger objects give no extension. Inner fields 2000 and 2000 red. V. $\frac{1}{2}$ part. (C., 1940.)

Interpretation.—The character of the field changes, especially the presence of nerve fibre bundle defects which extend across the vertical meridian, together with the absence of true hemianopic features, indicates the nerve as the site of the interference. In the rare cases in which indications suggestive of hemianopia are found, careful examination by quantitative perimetry is necessary to show whether signs of tract or chiasmal interference are really present, or whether the condition is merely one of homonymous or bitemporal depression simulating true hemianopia only in position and shape. Uhthoff, for example, in illustrating a case of apparent symmetrical inferior hemianopia, points out that the condition is really a peripheral shrinkage of each field, and suggests that in other cases the hemianopia may be due to a complication. The point is also discussed by other observers (Fuchs, Langenbeck, Paton), and it would seem to be advisable to regard the existence of true hemianopia as a purely tabetic manifestation with some reserve until more detailed evidence is forthcoming as to the exact nature of the field changes and the absence of any basal complications in such cases. The existence of genuine hemianopic changes would show that the lesion may begin in the tract or chiasma.

Similarly, with regard to the presence of central scotoma in uncomplicated tabetic atrophy, a closer analysis is required, not only of the scotoma, but of the whole field before the true relation of the defect to the tabetic process can be established. A condition of this kind would bring the patient quickly to the oculist, yet the development of perimetry and of neurological diagnosis has not increased such observations. Uhthoff never found central scotoma as an isolated field change, but nearly always associated with some peripheral depression, and it is probable that a more exact analysis would have shown that such central scotomata were really portions of sector defects whose intensity varied at different parts. There is no clear evidence of isolated involvement of the macular fibres, and in reported cases the characters of the defect have not always been sufficiently closely examined to exclude other conditions. The only instance I have personally seen was a tobacco scotoma. (*See also* Paton, 305.)

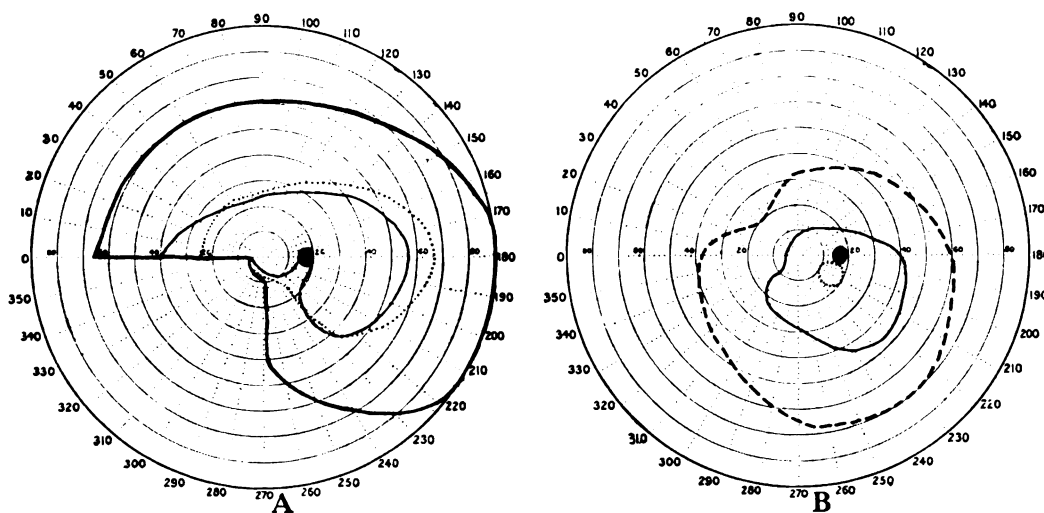


FIG. 134.—TABES. (FROM ROENNE, 327.)

To show proportion and disproportion between the fields for $\frac{1}{3}$ white, and $\frac{1}{3}$ red. In A these fields are proportionately altered, in B disproportionately. White, thin continuous line: red

Central scotoma, therefore, unless there is good reason to the contrary, should be regarded as a complication of tabetic atrophy. It may occur in other syphilitic affections of the optic nerves associated with atrophy.

Wilbrand and Saenger attribute field changes with rapid loss of central vision to a lesion in the anterior end of the nerve, since here the macular fibres lie peripherally, and peripheral loss with relative retention of central vision to a more posterior lesion, the macular fibres being affected late on account of their central position.

For the most part the disease affects the nerve, with possible unusual cases in which it originates in the chiasma or tract. The first type of field change indicates that the whole cross-section of the nerve may be fairly uniformly affected, the second that more or less sharply demarcated parts may be attacked. The significance of the field changes in regard to the pathological process is not clear. Paton regarded the general depression of the field as indicating a direct toxic action on the nerve fibres, and the more demar-

cated defects as the secondary results of localised chronic interstitial inflammation. On the other hand, the general depression of the fields with loss of colour vision in advance of that for white—disproportion—only indicates diffuse partial damage to the fibres, and does not seem necessarily to indicate direct toxic action as against vascular impairment. The different field defects may equally well be explained by diffuse or patchy interstitial disease of different degrees of severity, especially if, as is probable, the obliteration of minute blood-vessels plays an important part.

Diagnosis.—In the majority of cases, at the time when the patient presents himself on account of visual failure, the clinical picture is sufficiently developed to make diagnosis easy without a very close examination of the fields. When the visual failure precedes the general symptoms by a long interval the importance of the perimetric evidence is increased, especially as the ophthalmoscopic appearance of simple atrophy may be produced by other causes. The history of onset and the gradual relentless progress

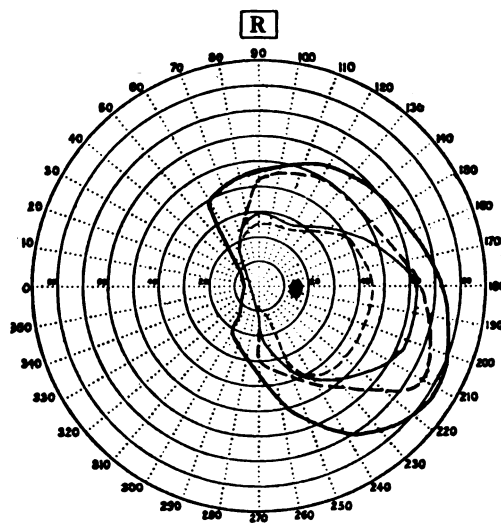


FIG. 135.—TABES.

Right field for 33° , 33° , 33° and 33° showing steep edge on nasal side and sloping edge on temporal side. V. = 3° . Left eye blind. (Gy., 1922.)

of the defects offer helpful indications, and diagnostic difficulties are less likely to arise if the case is seen early and observed over a period. It is noteworthy that at the time when visual failure is first complained of, the optic discs are practically always already pale. The pallor often commences before any functional defect can be detected except by the most careful quantitative testing.

In cases of suspected tabetic atrophy which appear to be unilateral, it is essential to test the apparently normal eye with small visual angles; the examination of central vision only is insufficient. Hemianopic defects require careful scrutiny; in tabetic atrophy they have the characters of sub-geniculate hemianopia, irregularity and incongruity with sloping edges, while the preserved portions of the fields may show asymmetrical changes indicating lesions of both nerves. In the progressive bilateral simple atrophy of chiasmal interference due to tumour, the type of hemianopia is so charac-

teristic that a misdiagnosis can hardly be made even on the perimetric evidence alone. When atypical defects such as hemianopia or central scotoma are present, the characters of the defects should, therefore, be minutely examined, and the possibility of complications, such as intracranial disease or toxic amblyopia, should be considered before admitting such changes as features of pure tabetic atrophy. Bilateral atrophy with field changes resembling in some respects those of tabetic atrophy may be present in certain cases of retrobulbar neuritis, cerebral syphilitic disease, or multiple sclerosis, and in some cases of toxic amblyopia due to certain arsenic compounds, but the onset and course of the defects, their relationship to the ophthalmoscopic appearances, and the general symptoms are different.

Bollack *et al.* (39) found concentric contraction of the fields in 23 per cent. of their collected cases of optico-chiasmic arachnoiditis.

Prognosis.—The presence of proportion or disproportion between the fields for white and colour is of value in indicating the relatively stationary or actively progressive nature of the conditions. Progress may, however, be variable, and both remissions and exacerbations may occur. Apart from the presence of disproportion or proportion the different types of field defect do not affect the immediate prognosis. As a general rule the worse the fields the worse is the expectation of retention of vision.

According to some observers indications with regard to treatment may be obtained from the fields. Behr (27), following Wilbrand, finds that energetic antisymphilitic treatment is contraindicated in three types of field :—

1. Impairment of central vision ; early failure for colour with normal or nearly normal boundaries for white.
2. Extreme concentric contraction with boundaries for white and colour falling together ; normal or nearly normal central vision.
3. Slight field changes which affect white rather than colour perception ; normal or nearly normal central vision ; already well developed pallor of the optic disc ; pronounced subjective light sensation. (*See also* Zimmermann (486).)

On the other hand, Igersheimer (197), with Uhthoff, is sceptical as to the results of treatment, one way or the other, whatever the state of the fields may be.

In any case it seems hardly justifiable to attribute further loss of vision, even if accelerated, to the treatment employed, especially when the field changes indicate a progressive type of atrophy. My experience indicates that the state of the fields need not be permitted to influence the treatment, which, in any case, should be conducted with care and prudence as regards dosage of arsenicals.

II

PRESSURE

The pressure group includes those cases in which the conduction interference depends upon local pressure on or proximity to the nerve such as is produced by neoplasms or other swellings, whether external to the nerve or in its substance or sheath. This cause may operate anywhere in the visual path, and as regards the optic nerve, affects most commonly the intracranial part on account of its anatomical relationships.

C.P.

o

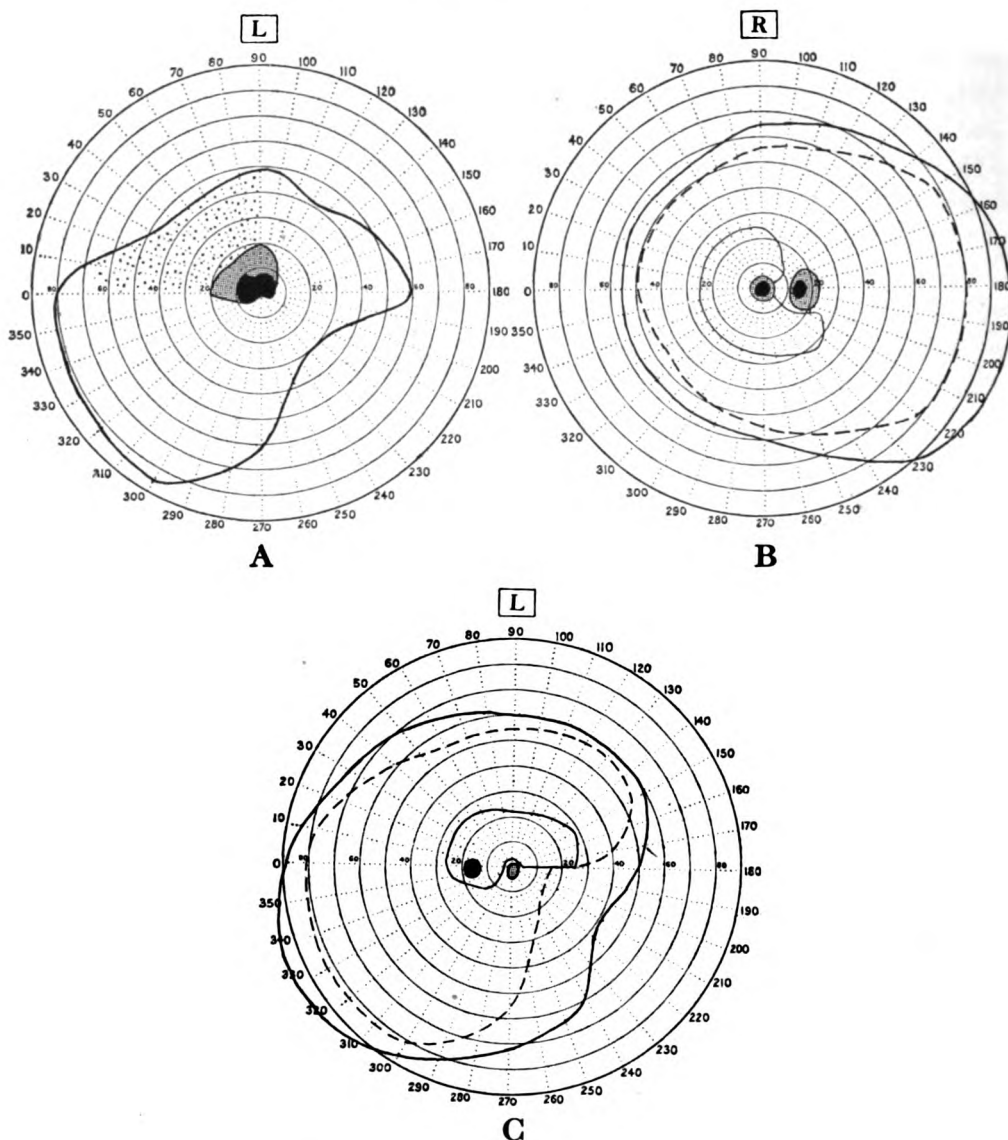


FIG. 136.—PRESSURE INTERFERENCE WITH THE OPTIC NERVE.

- A. Defect in upper temporal quadrant increasing in density towards centre. Pressure on left optic nerve at foramen. $3\frac{5}{30}$. V = Fingers at 1 m. (F., 1921.)
- B. Central and pericæcal scotomata in a case of encapsulated intra-orbital tumour. The pericæcal defect is probably mainly due to œdema of the optic disc. V. = Fingers at 1.5 m. Objects $3\frac{3}{30}$, $3\frac{1}{30}$; $2\frac{5}{30}$, $2\frac{3}{30}$. (W., 1916.)
- C. Pressure on left optic nerve from sphenoidal tumour. $3\frac{5}{30}$, $3\frac{2}{30}$; $2\frac{5}{30}$. Red $1\frac{0}{30}$ showed a large central defect breaking through in lower nasal quadrant. (O'D., 1924.)

The field changes are, as a rule, wide sector depressions of irregular quadrantic or hemianopic shape producing different forms of vertical or horizontal hemianopia, or defects which tend in that direction. One field only is affected at first, but both may become involved, and in the latter case the hemianopia may be bitemporal or horizontal, or possibly binasal, but is never homonymous. The internal isopters are affected before

the periphery and recover later on removal of the pressure. Central scotoma is frequent. Sooner or later the defect crosses the vertical meridian of the field, and in some cases a real chiasmal bitemporal hemianopia may develop if the lesion extends backwards to the chiasma.

In binasal hemianopia one field is affected before the other. A scotoma may develop but the field changes do not begin with bilateral central scotomata. The field defects may recover or remain stationary or progress to complete blindness. Central vision is nearly always reduced and there is often associated depression of the temporal fields. Other symptoms are usually absent.

Annular scotoma has been reported by Lillie and Adson in two cases. In each there was an enclosed central scotoma.

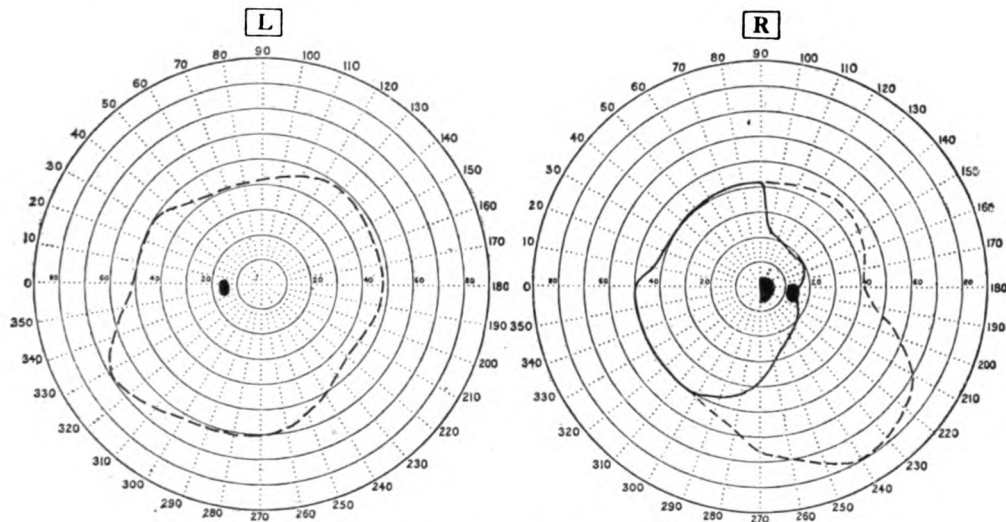


FIG. 137.—PRESSURE INTERFERENCE IN FRONT OF CHIASMA.

Right field shows temporal defect and temporal hemianopic scotoma. V. $\frac{6}{80}$. Objects $\frac{30}{30}$, $\frac{5}{30}$. Left field at this date practically normal for $\frac{3}{30}$, and no defect could be elicited with other tests. V. $\frac{8}{80}$. (Jn., 1921.)

The intensity of the defect varies, it is often greater centralwards so that a central or centrocaecal scotoma is present. The slope of the edge also varies at different parts and at different times. Sometimes the peripheral depression is minimal or even apparently absent, and the scotoma may show no hemianopic features.

The onset is usually gradual and the patient seeks advice when central vision becomes affected. As the invasion of the fixation area is easily noticed, while peripheral failure tends to escape observation, the patient may give a history of comparatively rapid onset. In some cases the onset actually is rapid, and progress may be rapid or slow according to the nature of the lesion. The peripheral depression becomes more intense and the affected parts of the field become blind. Ultimately the whole field may be lost.

The changes are progressive although sometimes varying, and the tendency to recovery is pronounced. Recovery occurs rapidly when the pressure is relieved, unless it has acted for a long time.

Interpretation.—A defect confined to one field indicates interference with one nerve; when both fields are involved the defects are usually bitemporal and indicate a source of pressure lying between the nerves. When a temporal hemianopia is present in one field with integrity of the other, the apparently normal field should be subjected to the most rigorous examination before deciding that it really is normal, and it should be carefully watched as a tumour near the chiasmal termination of one nerve, whether on its medial, or its lateral side, is likely to cause changes in both fields sooner or later. Diagnosis should therefore be provisional in the first instance. Involvement of the chiasma, which may occur in such cases, is indicated by the development of the characteristic field changes of bitemporal hemianopia, though it may be difficult at first to decide whether these are due to interference with the medial sides of both nerves in the anterior chiasmal angle or to interference with the anterior part of the chiasma itself as

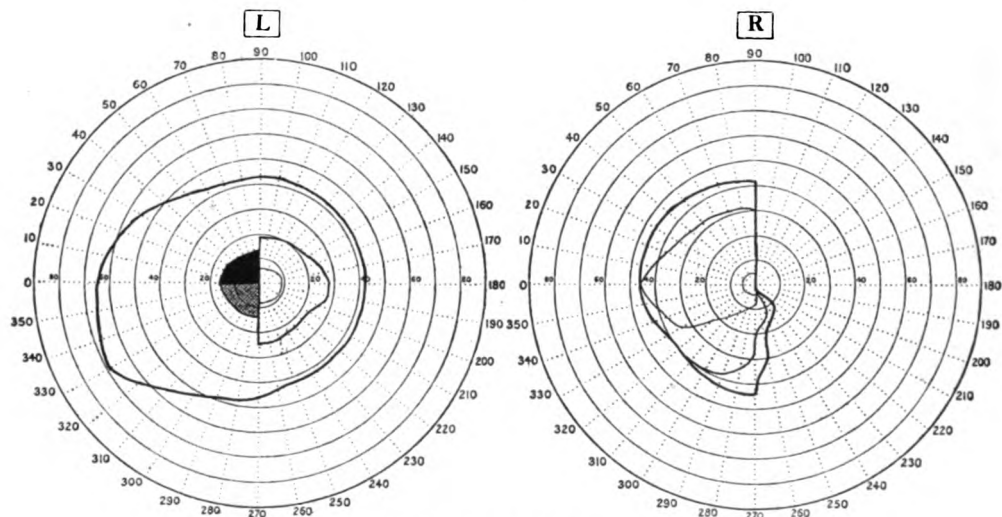


FIG. 138.—SAME CASE AS FIG. 137 TWO YEARS LATER.

Right field shows absolute temporal hemianopia. Objects $\frac{5}{30}$, $\frac{2}{30}$, $\frac{5}{30}$ and $\frac{3}{30}$. V. $\frac{6}{60}$.
 Left field shows great depression and temporal hemianopic scotoma. Objects $\frac{5}{30}$, $\frac{2}{30}$, $\frac{5}{30}$. V. Counts fingers at 1 m. (Jn., 1923.)

the crossed fibres are affected in both cases. The possibility of bilateral defects being due to interference with the chiasmal termination of one nerve has been referred to (pp. 84, 171). Binasal hemianopia indicates bilateral interference with the uncrossed fibres of the optic nerves, the crossed fibres being intact. The lesion appears to be as a rule at the outer side of the termination of each nerve as it joins the chiasma, possibly due to the proximity of the carotid arteries.

In persistent unilateral cases the lesion may be placed more anteriorly in the nerve, especially when hemianopic features are not prominent or are absent. The rate of advance in the field changes corresponds approximately to the rate of increase in the pressure. When the pressure is active there is also considerable general depression with pronounced central scotoma and disproportion between the fields for white and colour.

Stewart and Riddoch have shown that pressure upon the spinal cord produces its effects by causing venous congestion and arterial ischæmia rather than by compressing the nerve fibres themselves, and it may be assumed that pressure upon the optic nerve and other parts of the visual nerve path acts in the same way. The pathological lesion has been found by Roenne (353) to be similar to that present in retrobulbar neuritis and, as the nerve need not be actually squeezed, he prefers to regard the condition as a neighbourhood (*Nachbarschaft*) rather than a compression effect.

Diagnosis.—From the clinical point of view it is important not to confuse pressure interference with retrobulbar neuritis. The characters described, together with the history and course of the field changes, considered in association with the evidence from other sources, often enable the correct diagnosis to be made or, at least, guide the observer in the proper direction.

In unilateral cases without obvious hemianopic features a local orbital cause affecting the intraorbital portion of the nerve should be suspected. Orbital swellings usually present little difficulty, but when the apical region is involved there may be proptosis only, and the presence of a swelling within the orbit may be doubtful. Here tumour of the nerve sheath should be thought of, and careful radiographic examination of the optic foramen and its neighbourhood is indicated.

The intracranial part of the nerve may be affected by meningeal tumours growing from the region of the tuberculum sellæ or by glioma of the nerve itself. The former tend to involve the medial side of each nerve, one after the other, and the latter may be either uni- or bilateral. Glioma of the nerve is a rare condition: the field changes consist of a slowly progressing depression and constriction and are not in any way specially characteristic. The field changes due to a prechiasmal tumour between the optic nerves often do not show the regular orderly progress characteristic of typical chiasmal pressure interference from below, but tend to exhibit more irregular defects though still definitely of bitemporal hemianopic type. Blindness of one eye with temporal hemianopia in the other may result. The course is usually long and progress slow but certain. Occasionally remissions or alterations in the defects may occur which must not be allowed to influence the diagnosis unduly. Cushing believes that in these cases the nerves suffer more than the chiasma since his post-operative results relatively frequently showed restoration of vision in the eye, which had previously had temporal hemianopia, the blind eye remaining blind, whereas in chiasmal interference restoration of both nasal fields (uncrossed fibres) would be expected.

Interpeduncular tumours or tumour or abscess of the frontal lobe, or meningioma of the cribriform plate, may affect this part of the nerve without involving the chiasma. In frontal lobe cases, as Foster Kennedy and Uhthoff have shown, there may be papilloedema on both sides or papilloedema on one side and pallor of the disc on the other. Central scotoma is found on the side of the pallor if this is present, the other field shows no change or only slight peripheral restriction with some enlargement of the blind spot—the usual signs present in papilloedema due to increased intracranial pressure.

The Foster Kennedy syndrome * consists of primary optic atrophy with central

* Originally noted by Paton (304).

scotoma on one side and papilloedema on the other. Variations may occur. The syndrome is common in meningioma near the optic nerve and, when combined with anosmia and mental disturbance, is almost pathognomonic of meningioma in the region of the cribriform plate.

Lillie found a central or centrocæcal scotoma in ten out of fourteen cases of basal frontal lobe lesion. The scotoma was always on the side of the tumour. Of the other four cases three showed homolateral blindness complete or nearly complete, and one with bilateral lesions had bilateral blindness. The scotoma is due to the direct or indirect effects of pressure by the enlarged frontal lobe upon the subjacent optic nerve, and disappears if the pressure is relieved.

Bilateral inferior altitudinal hemianopic changes have been ascribed to elevation of the nerves pressing them against the sharp upper margins of the optic foramina which are overlaid by a stiff fold of dura mater.

Pressure effects due to indentation of the nerves by hardened arteries have been described, but do not appear to be of much practical importance excepting in so far as they may explain some obscure cases of simple atrophy in elderly persons who have arterial disease. Knapp (221, 222) has referred to the value of field examination especially in relation to the course of the changes in the differential diagnosis between these cases and glaucoma. An element of vascular obstruction may also be present (p. 202). More important is the constriction of the upper surfaces of the nerves by the circulus arteriosus or by the upper edge of the optic foramen when they are forced upwards by a subchiasmal tumour. One or both fields may show defects of inferior altitudinal hemianopic type, and signs of chiasmal interference are also present.

Binasal Hemianopia

Binasal hemianopia is believed to be due to distension of the third ventricle from increased intracranial pressure causing the anterior angles of the chiasma to be displaced laterally and thus producing compression of the lateral sides of the terminations of the nerves against the carotid arteries. Hence its occurrence as an accompaniment of postneuritic atrophy (Cushing and Walker, 73). According to Lutz (267) it is especially liable to be produced in connection with tumours of the wall of the third ventricle or of the choroid plexus, or subtentorial tumours. It may also be caused by hardening of the carotid arteries, or aneurisms of both carotids, symmetrical gummata, tabes and some other conditions. It begins in the lower quadrants and progresses slowly. It is never initiated by central scotoma and never appears suddenly. Binasal hemianopia is, of course, not a true hemianopia in the sense of a bilateral field defect due to a single lesion in the visual path, and is usually irregular and unequal or associated with changes in the temporal fields. Beyond indicating that the site of interference is subchiasmal, it is not of great value in diagnosis.

Theoretically binasal hemianopia might be expected to result from symmetrical lesions on the lateral side of each tract. There is no evidence of such causation.

Prognosis.—Pressure defects show a pronounced tendency to recovery if the cause is removed before permanent damage is done and vision may be restored even

after the lapse of months. The prognosis is therefore good if the pressure can be relieved, even though considerable pallor of the disc is present, while the degree of recovery depends upon the stage at which the interference is removed. The fields should always be tested for colour, as the presence or absence of disproportion influences the prognosis materially.

III. PLEROCEPHALIC ŒDEMA (ŒDEMA DUE TO INTRACRANIAL PRESSURE "CHOKED DISC" OR "PAPILLOEDEMA") AND "POSTNEURITIC" ATROPHY

The field changes produced by œdema of the optic nerve due to increased intra-

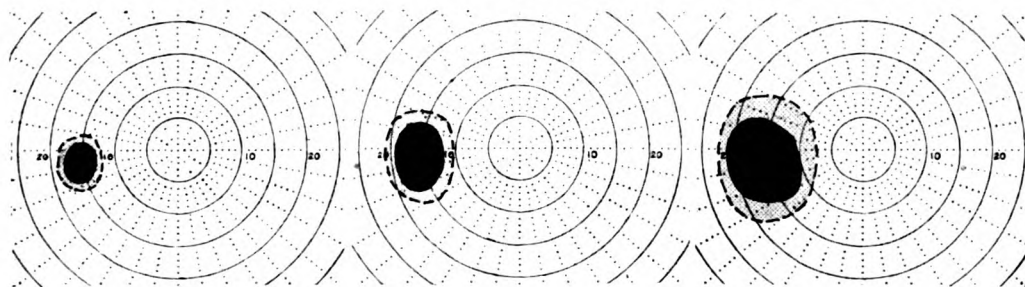


FIG. 139.—ŒDEMA OF DISC FROM INCREASED INTRACRANIAL PRESSURE.

Objects $\frac{300}{2000}$, $\frac{2000}{2000}$. 1924, July 25th, October 12th, and November 9th, from a case of brain tumour. Vision $\frac{8}{8}$ throughout. (R., 1924.)

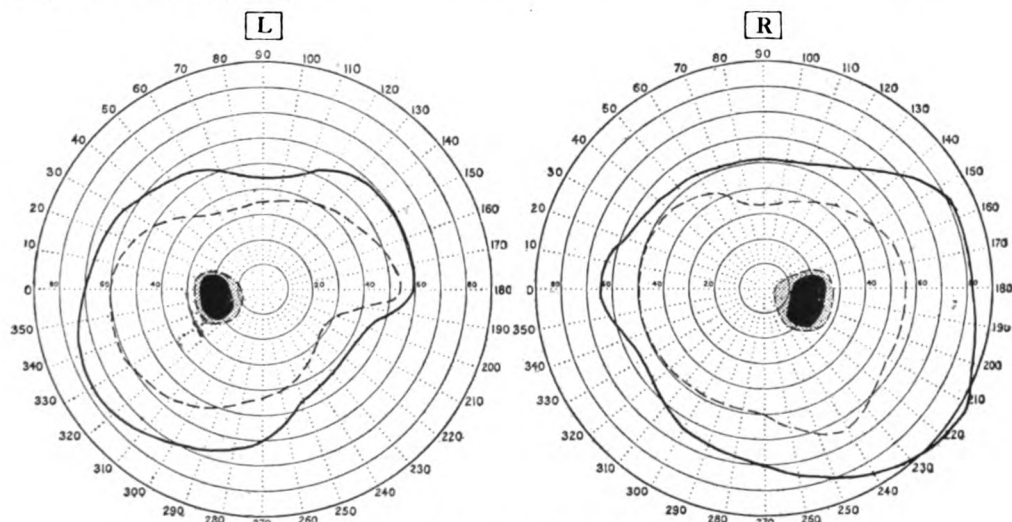


FIG. 140.—PLEROCEPHALIC ŒDEMA IN BRAIN TUMOUR.

Greatly enlarged blind spots with defect extending towards fixation point. Relative central scotomata (not charted) due to macular œdema were also present. Commencing atrophy on left side indicated by peripheral contraction. R.V. $\frac{1}{2}$ part. L.V. $\frac{3}{4}$ part. Objects $\frac{330}{330}$, $\frac{330}{330}$: $\frac{2000}{2000}$, $\frac{2000}{2000}$ (blind spots). (B., 1925.)

cranial pressure from any cause, consist firstly of those due solely to the œdema, and secondly of those due to the ensuing atrophy. Complications may also be present in the form of defects due to direct interference with the visual path by the cause which produces the increased intracranial pressure, or may rise from other sources.

"Enlargement of the blind spot" has been described by De Schweinitz as occurring before ophthalmoscopic evidence of papilloœdema is present. Personally, I have found

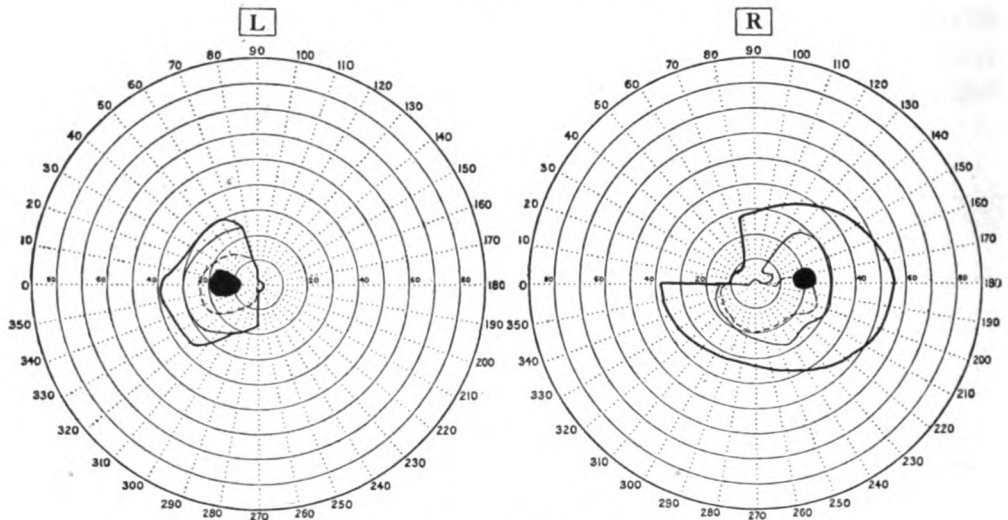


FIG. 141.—“POSTNEURITIC” ATROPHY (Atrophy following pteroccephalic oedema).

Binasal defects with great peripheral depression. R.V. $\frac{6}{18}$. Objects: $\frac{20}{330}$, $\frac{20}{1000}$, $\frac{10}{1000}$. L.V. $\frac{5}{60}$. Objects: $\frac{10}{330}$, $\frac{20}{1000}$. (C., 1911.)

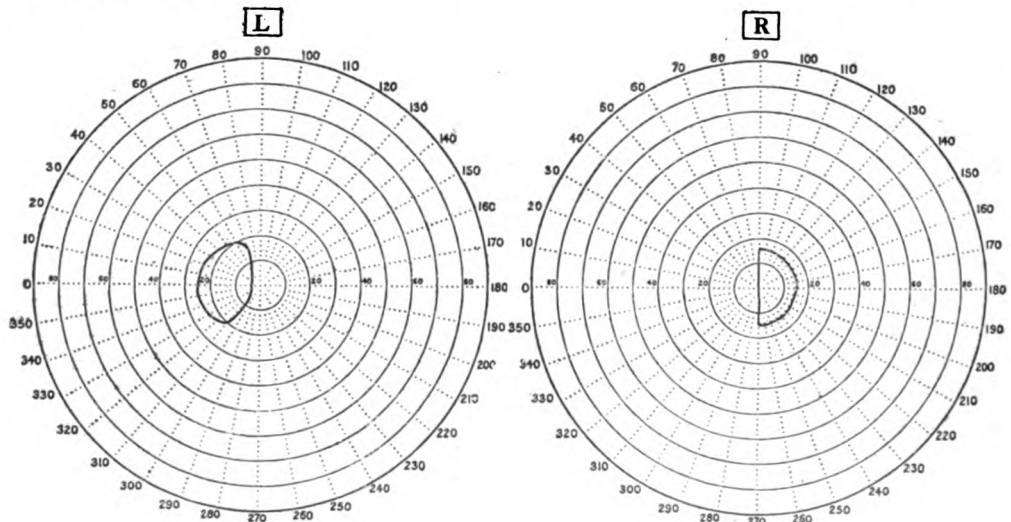


FIG. 142.—POSTNEURITIC ATROPHY, CAUSE UNKNOWN.

Binasal hemianopia with great depression of the fields. V.O.D. = H.M. V.O.S. = Fingers. The nasal margin of the right field was probably not so straight as shown. Objects R. and L. $\frac{20}{330}$. (C., 1916.)

the fields normal until the swelling of the nerve head is definite. At this stage a pericæcal defect develops which begins as a slight enlargement of the normal amblyopic zone and enlarges to a scotoma extending for several degrees round the blind spot and half way or even farther towards the fixation point. Its intensity diminishes towards its periphery, where it slopes with a varying degree of steepness into the unaffected part of the field. The onset is usually gradual, but in certain cases may be rapid, and the area of the defect corresponds to the degree of oedema of the nerve head and surrounding retina, though not quite so extensive. If the oedema extends into the macular area the

changes there soon produce slight depression of central vision in the form of a small central relative scotoma, which is more easily demonstrated at first by colour tests and presents the relative blue-blindness characteristic of a retinal outer layer disturbance. A relative centrocaecal defect may result. Apart from this condition central vision is not affected by uncomplicated plerocephalic oedema without atrophy.

As atrophy sets in the field becomes depressed and concentric contraction appears. The internal isopters are less affected than those which lie peripherally, so that peripheral vision fails more rapidly than central vision (apart from the macular oedema referred to above), and in the late stages a patch more or less central in position or round the blind spot is the last to disappear. In the latter case the earlier loss of the nasal fields may produce a resemblance to binasal hemianopia.

Complications due to localised interference consist for the most part of hemianopic changes or unilateral central scotoma. Uhthoff found homonymous hemianopia in 2·7, heteronymous (temporal) hemianopia in 1, central scotoma in 2, and ring scotoma in 0·3 per cent. of his cases. Nerve fibre bundle defects have been noted by Roenne, von Szily and others, and are ascribed by the former to accidental special implication of a bundle at the disc margin.

The defects due to atrophy following the oedema and those due to different kinds of local interference may be present together, and ultimately, if the process is continued, the local interference changes may become difficult to recognise owing to the general depression of vision and the contraction of the fields.

Interpretation.—The pericaecal scotoma may be regarded as depending upon the enormously increased thickness and opacity of the nerve fibre layer immediately around the optic disc. At the edge of the scleral opening the retina is also compressed, crumpled and pushed away, so that the area devoid of percipient elements becomes increased. The central scotoma is due to the macular oedema, and in severe cases may merge into the pericaecal defect. These changes are merely a result of the swelling and congestion and have no special significance; it is important not to mistake them for visual loss due to direct interference with the nerve path or to commencing atrophy. The subsequent depression and peripheral contraction are due to interstitial changes in the nerve, which, while present in the whole cross-section of the nerve, are, as a rule, more severe peripherally, and spread inwards causing a diminution in the blood supply to the fibres. Central scotoma, other than that caused by retinal oedema, or hemianopia point to complications, and their characters indicate the situation of the interference with the nerve path.

Diagnosis.—The existence of oedema of the nerve and the state of the macular area are ascertained by the ophthalmoscope and the function of perimetry is the detection of direct interference with the visual path and of the first signs of commencing optic atrophy. It is not always possible to distinguish by the ophthalmoscope plerocephalic oedema from the oedema produced by a local optic nerve lesion, but mistakes are likely to be avoided if it is remembered that in the former central vision is never affected, except by a complication, until a late stage is attained, while in the latter central vision is almost always involved from the beginning. Exceptions to this rule are rare.

and usually easily diagnosable in other ways (*e.g.*, œdematous disc with nerve fibre bundle defect in the field in choroiditis juxta-papillaris).

The fall in vision due to macular changes is never severe and presents the distinctive retinal characters already referred to. It should not be confused with that produced by commencing atrophy, and in this differentiation perimetry is of assistance as visual loss due to atrophy appears first in the peripheral zone of the field where it should be sought while central vision is still good. Careful examination by perimeter and screen, using visual angles which are sufficiently small, is required in order that the earliest signs of atrophy may not escape detection. If macular changes, direct interference, and commencing atrophy are all present together, care is required in attributing each feature to its proper cause, but no great difficulty is introduced, as a rule, until the atrophy has progressed sufficiently to mask the other conditions.

Central scotoma in one field in cases of choked disc has already been mentioned as a pressure interference, it has also been recorded as resulting from combined retrobulbar neuritis and meningitis. The significance of hemianopia will be referred to later.

Prognosis.—The ophthalmic surgeon is sometimes asked how long it is safe to postpone a decompression operation, and in this connection perimetry is a valuable complement to ophthalmoscopic examination. Once visual loss due to post-œdematous atrophy has commenced, it may advance rapidly, and after a certain stage has been reached decompression often fails to prevent further progress to complete blindness. Early signs of peripheral failure should be sought as soon as the patient comes under observation, and the examination should be repeated at intervals using delicate tests. The ophthalmoscopic appearances and the state of central vision alone do not give the earliest indications of commencing atrophy.

In the absence of complications the prognosis as to vision is excellent if decompression is done in time.

IV. VASCULAR DISEASE

Occlusion of vessels in the nerve may occur in arterio-sclerosis or syphilis, leading to a greater or less extent of damage to the fibres. Field changes resembling those of retrobulbar neuritis ensue and general depression, wide sector defects which may involve the fixation area, and central scotoma may be present, or general depression without other discoverable features. The visual loss may be very severe. The course of the field defects corresponds to the onset of the obstruction. If this is sudden the case may resemble one of retrobulbar neuritis, both as regards field changes and ophthalmoscopic appearances (œdema of nerve head), if the onset is gradual the conditions may suggest a simple atrophy. The field changes, together with the ophthalmoscopic appearances and other signs of vascular disease in the patient, who is usually elderly, form the basis of the diagnosis. A high blood pressure is suggestive, but this is not always present. In the type with gradual onset the field changes may in some ways resemble those of chronic glaucoma, but are distinguished by differences in course. Increased intraocular pressure is absent but some cupping of the disc may be found (Knapp 221, 222). As the blood supply of the nerve fibres may be destroyed beyond restitution the prognosis

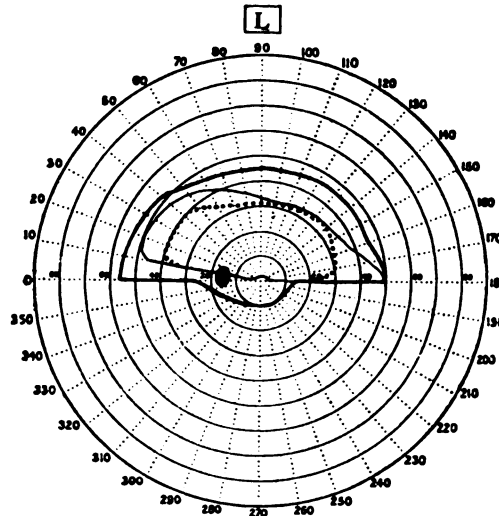


FIG. 143.—VASCULAR DISEASE AFFECTING THE OPTIC NERVE.

Wide sector defect with central scotoma and peripheral depression in retained field. Objects $\frac{30}{330}$, $\frac{5}{330}$; $\frac{10}{330}$ red. Vision = Fingers at 1 m. (J. S., 1912.)

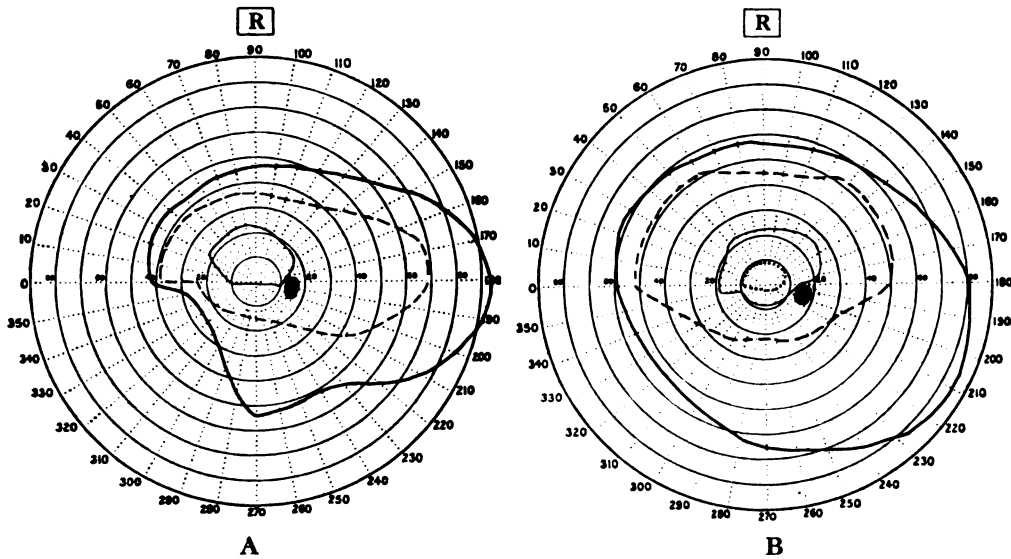


FIG. 144.—INJURY.

A. Five weeks after injury. V. = $\frac{6}{24}$. Objects $\frac{20}{330}$, $\frac{30}{330}$; $\frac{6}{330}$. (S., 1924.)

B. Another case, ten weeks after injury. V. $\frac{6}{24}$. Objects $\frac{30}{330}$, $\frac{10}{330}$; $\frac{20}{330}$ white and $\frac{10}{330}$ red. (P., 1923.) Note horizontal hemianopia for small objects in both cases.

is bad and permanent loss of vision results, though some recovery may take place in the earlier stages.

Cases have been reported by Beselin (35) and Uhthoff (430).

V. TRAUMATISM

The nerve may be injured as a result of violence, with or without a fracture through the optic canal. The most common cause is a blow on the temple often due to a fall

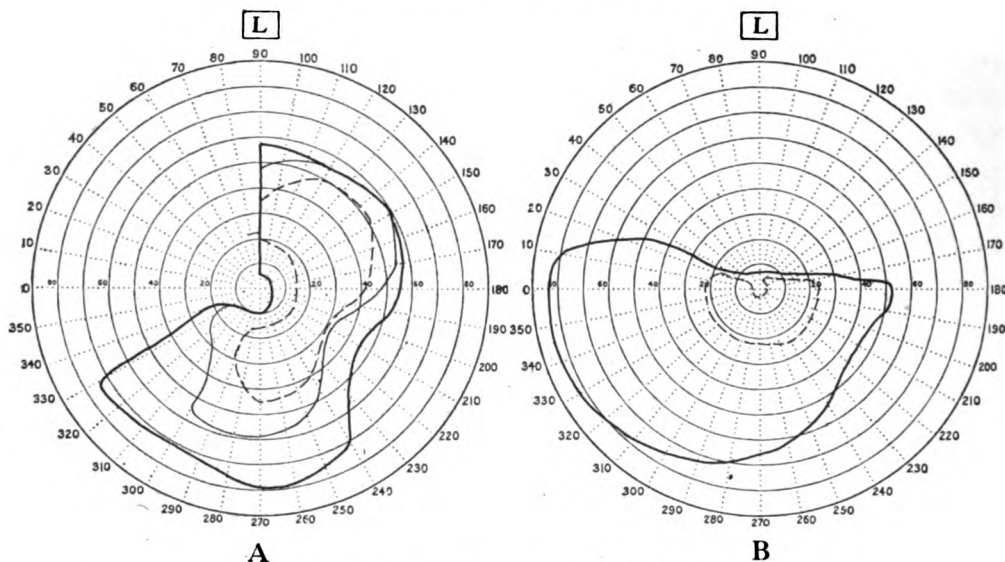


FIG. 145.—INJURY. MORE SEVERE CASES.

A. Objects $\frac{60}{330}$, $\frac{20}{330}$, $\frac{5}{330}$. Nine months after injury. V = Fingers. Temporal hemianopic defect. (H., 1923.)
 B. Objects $\frac{20}{330}$, $\frac{5}{2000}$. Blind spot not marked. Two months after injury. Superior hemianopic defect. V. $\frac{6}{80}$. (F., 1923.)

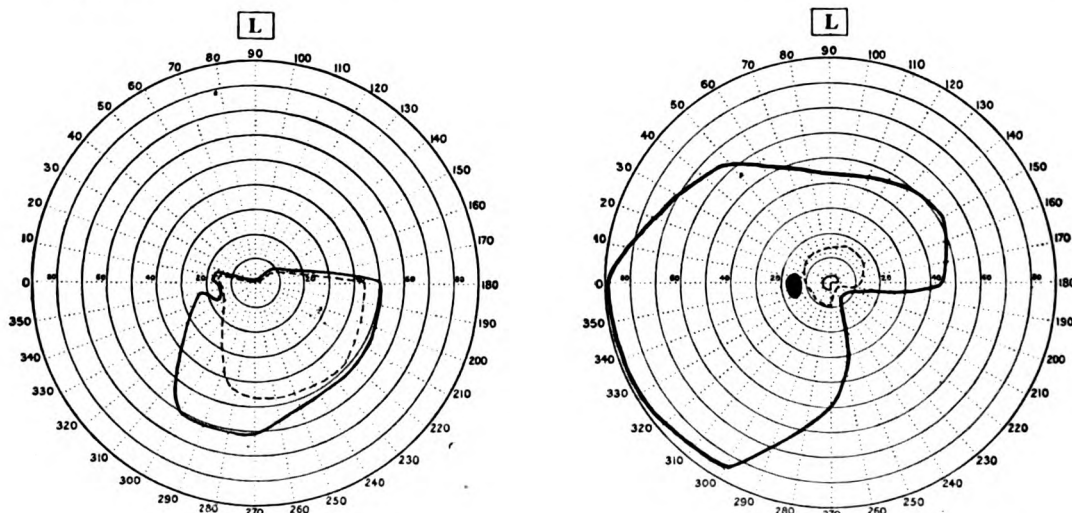


FIG. 146.—INJURY.

Blow on left forehead due to a fall of 25 feet. Loss of field except lower nasal quadrant. V. $\frac{2}{60}$. Objects $\frac{10}{330}$, $\frac{5}{330}$. (G., 1936.)

FIG. 147.—INJURY. MOTOR ACCIDENT

Fracture of base of skull. Loss of lower nasal quadrant of field of left eye. Objects, $\frac{10}{330}$, $\frac{10}{2000}$, $\frac{10}{2000}$: $\frac{20}{2000}$ red. V. $\frac{5}{6}$. (C., 1932.)

from a bicycle. There may be no detectable fracture of the skull and the most interesting cases are those in which the violence has been apparently very slight. Very frequently immediate, complete and permanent blindness of the eye on the injured side ensues. In other cases some recovery of vision occurs within the first few weeks, but any defects which remain at the end of a month or more are permanent. In slight cases almost complete recovery may result but some central depression of the field may remain,

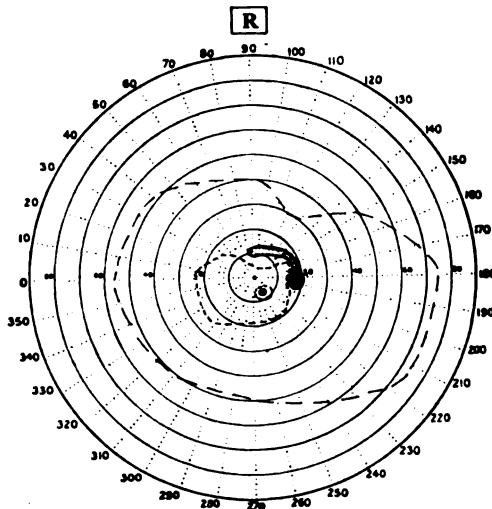


FIG. 148.—NERVE FIBRE BUNDLE DEFECT FROM INJURY.

Fall from bicycle. Indentation of upper temporal quadrant with nerve fibre bundle defect and minute central scotoma. Another small scotoma below fixation area. V. $\frac{1}{2}$. Objects $\frac{1}{2}$ 100, $\frac{1}{2}$ 100, $\frac{1}{2}$ 100 and $\frac{1}{2}$ 100. (B., 1936.)

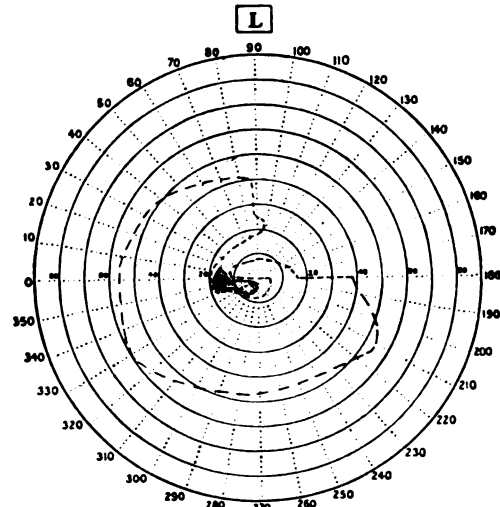


FIG. 149.—INJURY. NERVE FIBRE BUNDLE DEFECT.

Fall from motor cycle. Arcuate defect from blind spot into upper nasal quadrant. Centrocæcal scotoma. V. $\frac{1}{2}$. Objects: $\frac{1}{2}$ 100 blind spot: $\frac{1}{2}$ 100 scotoma: $\frac{1}{2}$ 100 peripheral field. (Bn., 1936.)

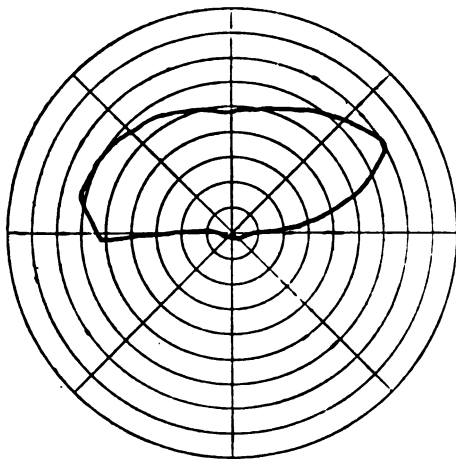


FIG. 150.—INJURY TO RIGHT OPTIC NERVE BY SHOT PELLET THROUGH UPPER LID.

$\frac{1}{2}$ 100. V. $\frac{1}{2}$. (W., 1941.)

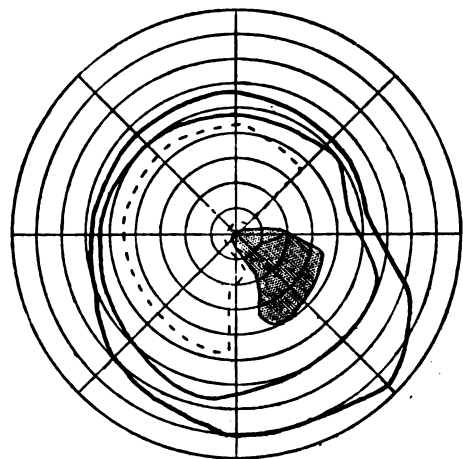


FIG. 151.—INJURY FROM FALL ON RIGHT TEMPLE.

Isopters for $\frac{1}{2}$ 100 and $\frac{1}{2}$ 100 slightly restricted on temporal side. Isopter for $\frac{1}{2}$ 100 shows extensive temporal defect. Large scotoma for $\frac{1}{2}$ 100. V. $\frac{1}{2}$. (M., 1938.)

together with pallor of the optic disc. The field shows depression and a more or less extensive sector defect which may occupy less than a quarter or more than half of its area. Only an eccentric patch may remain. Central, paracentral, or arcuate scotoma with intact or relatively intact peripheral field occasionally occurs. No special position or shape of the defect appears to be characteristic. The defects vary in intensity, but are usually absolute or severe with steep edges.

The nature of the field changes indicates that the lesion is in the nerve ; occasionally bitemporal hemianopia occurs pointing to damage to the chiasma. The partial recovery, leaving a permanent complete sector defect, suggests that the cause is not pressure due to blood effused into the sheath or direct damage to the nerve fibres, but probably the rup-

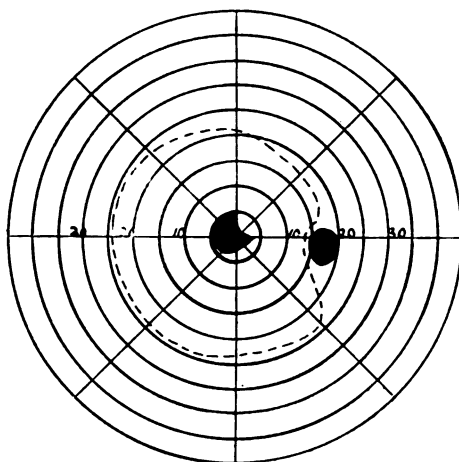


FIG. 152.--INJURY FROM FALL ON RIGHT SIDE OF HEAD.

Central scotoma with intact periphery. No fracture shown by X-ray. V. 60. Objects : 2000, 2480. (P., 1944.)

ture of vessels passing from the sheath into the nerve substance depriving the nerve fibres of their blood supply. The lesion is usually in the optic foramen. Lillie and Adson have recorded a case with ring scotoma following the development of callus after fracture through the optic foramen.

The examination of the fields may be of value in medico-legal cases, and some help in regard to prognosis may be obtained. It is unjustifiable to state that the field is normal, even though central vision and the peripheral limit of the field are not below normal standard, unless the positions of the internal isopters have also been examined and found to be normal. If disproportion is present and the edges of the defect are sloping, further improvement may be expected, but if the margins are steep and the white and colour fields in proportion not much more recovery will occur. Pallor of the optic disc appears in about three weeks and reaches its maximum in two months. Though the field defect may be limited to a quadrant the pallor affects the whole disc approximately uniformly.

CHAPTER X

CHIASMA

OUR appreciation of the field changes which are associated with lesions involving the chiasma is greatly facilitated by what is known of the fields in affections of the optic nerve. It is only necessary to apply the principles already studied to the special anatomical arrangements of the chiasmal fibres in order to understand easily the main characters of the field defects which depend upon interference at this point. Almost without exception these changes are so typical that they definitely indicate involvement of the chiasma.

On account of its situation the chiasma is especially liable to external pressure from tumours and other swellings. Intracellular and extracellular growths connected with the hypophysis and infundibulum, basal tumours of other kinds, aneurisms of the adjacent blood vessels, syphilis of neighbouring structures, and traumatism are among the conditions which may affect it. Multiple sclerosis, syphilis and glioma may also affect the chiasma itself. In all cases visual changes are produced.

The characteristic field defect resulting from chiasmal interference is bitemporal hemianopia, which in its typical form is found in cases of hypophysial tumour. Many varieties of bitemporal hemianopia used to be described until it was shown, originally by Josefson, that the so-called varieties are stages in a progressive process.

Two types may be recognised, the scotomatous and the non-scotomatous.

Scotomatous Type

The field begins to show depression in the upper temporal quadrant where the isopters therefore move towards the centre. While this failure is noticeable at the periphery in sufficiently advanced cases, if a medium size of test-object is used, and is, therefore, called peripheral indentation or "temporal slant" (owing to the loss of the normal outward convexity), it will be immediately recognised, if several internal isopters are examined whether with colour or small white tests, that practically the whole quadrant is affected even at a very early stage. The periphery may appear almost or quite completely normal for a test as small as $\frac{1}{330}$, while a definite upper temporal quadrant defect is present for $\frac{1}{2000}$. Sometimes, though a distinct scotoma may not be demonstrable, the more central isopters may show a more definite alteration than those which are normally more peripheral. In the earliest stages the only sign may be a slight upper temporal restriction of the isopter for $\frac{1}{2000}$, while colour perception in that quadrant, even for small objects, is merely dimmed. It may be necessary to increase the distance to more than 2 m. and to reduce the illumination in order to obtain evidence of hemianopia. In any case the earliest field changes will be found in the central area of the field and not at the periphery.

Towards the vertical meridian the edge of the depression is fairly sharply defined, and as the defect progresses the edge here becomes steep and ultimately perpendicular, but the vertical meridian is not crossed in the upper quadrant. Below, the depression

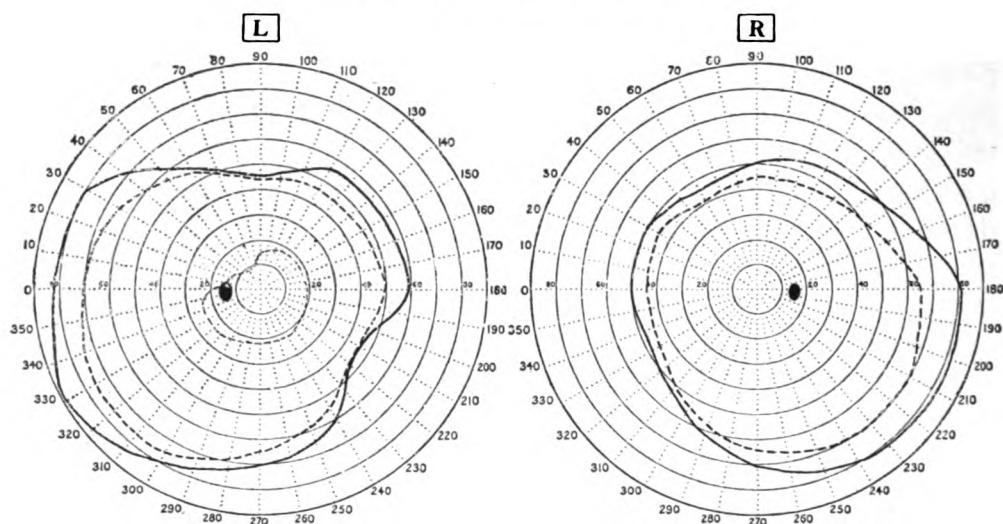


FIG. 153.—BITEMPORAL HEMIANOPIA. EARLIEST STAGE.

Objects 330° , 330° ; 200° . Characteristic change in central area of one field only. Right field showed no defect for white or colour. Acromegaly: eighteen months after onset of general symptoms. R.V. $\frac{5}{6}$. L.V. $\frac{5}{6}$. (P., 1923.)

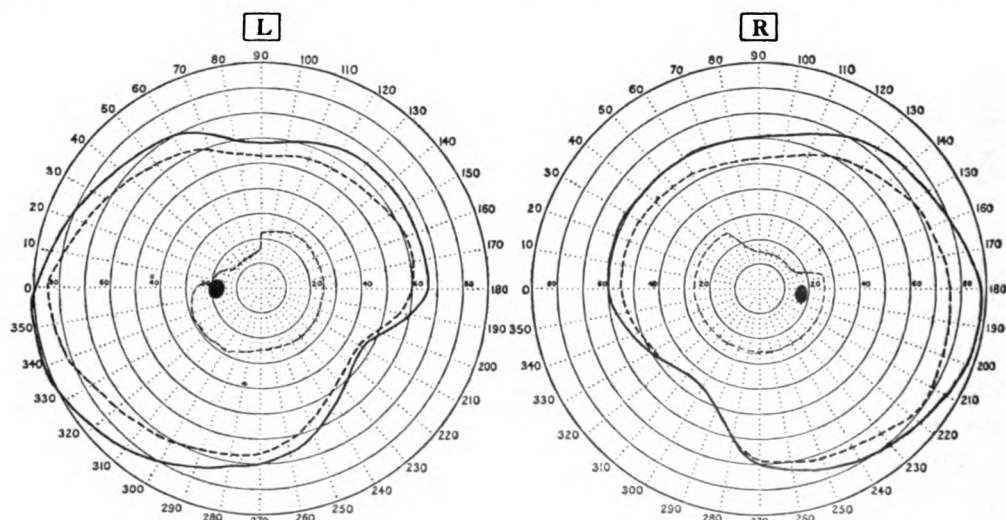


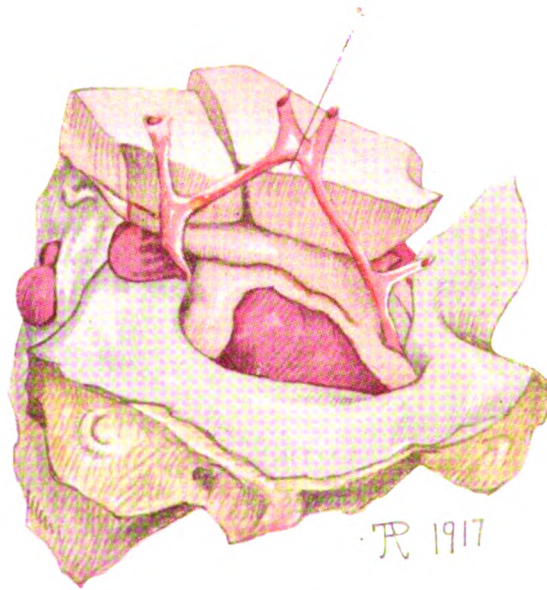
FIG. 154.—BITEMPORAL HEMIANOPIA. EARLIEST STAGE.

Objects 330° , 330° ; 200° . The left field also showed a slight relative defect for colour in the temporal outer quadrant. Note normal peripheral field. R.V. $\frac{5}{6}$; L.V. $\frac{5}{6}$. Nine months after onset of general symptoms: acromegaly. (C., 1922.)

slopes more or less into the still unaffected lower temporal quadrant except where there is a scotoma with a steep inferior margin.

About this time the apex of the upper temporal quadrant shows a scotoma bounded by the vertical and horizontal meridians of the field along which, especially the vertical, it is sharply defined. This scotoma enlarges upwards to meet the down-growing indentation and breaks through to the periphery. It also expands downwards into the lower temporal quadrant, giving the field an uncinatè or gourd-like outline, with a hook-shaped

PLATE IV.



TUMOUR WHICH CAUSED CHANGES SHOWN IN FIG. 156.

Note the separate anterior commissure of the chiasma, also the manner in which the tumour pierces the dura mater covering the cavernous sinus. Natural size.

[To face p. 209.]

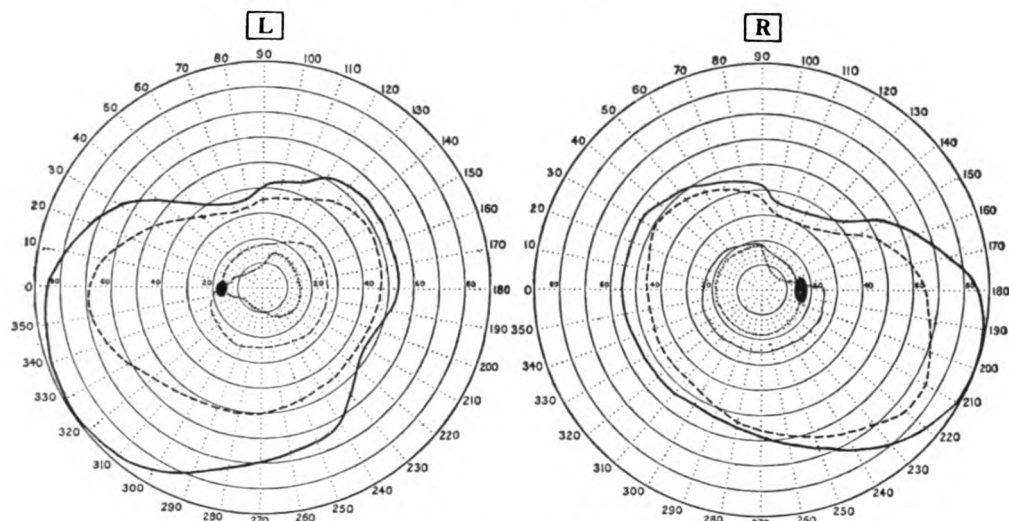


FIG. 155.—BITEMPORAL HEMIANOPIA. COMMENCING PERIPHERAL DEPRESSION.

Objects $330, 330; 2000, 2000$. Also 2000 red (dotted line). for colour than for white: temporal "slant" appearing. symptoms: acromegaly. (B., 1913.)

In the left field the central depression is shown better R.V., L.V. $\frac{1}{2}$. Five years after commencement of gland

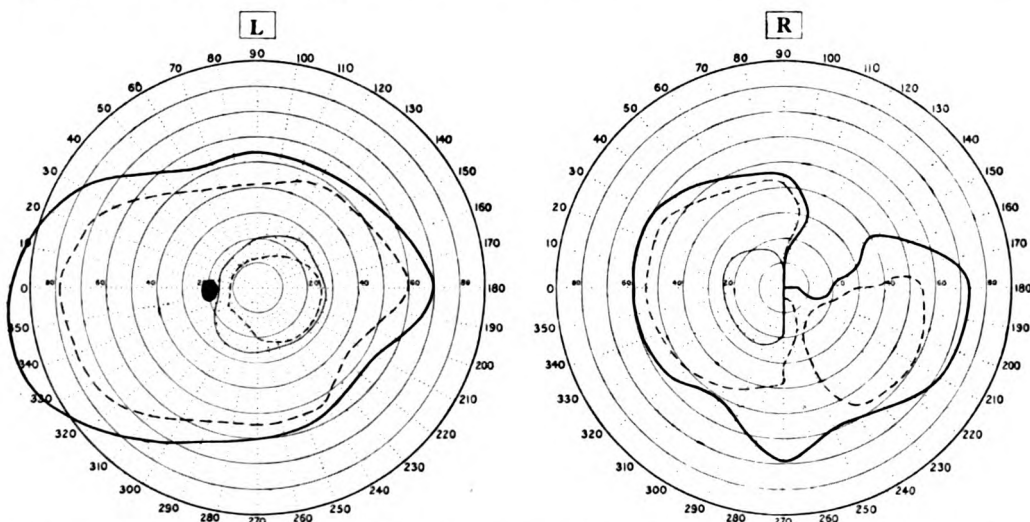


FIG 156.—BITEMPORAL HEMIANOPIA. EARLY CHANGES IN LEFT FIELD WITH ADVANCED STAGE IN RIGHT

Objects $330, 330; 2000, 2000$. Right field shows loss of upper quadrant splitting off of temporal island, and complete hemianopia for a small object. Acromegaly: five years' duration. R.V. $\frac{1}{2}$. L.V. $\frac{1}{2}$. (H., 1917.) Interference at right anterior angle of chiasma at medial side. (Trans. Ophth. Soc. U.K., XL., 198.)

process extending temporally and upwards below and around the temporal side of the blind spot. The lower quadrant then fails peripherally below, its isopters become indented, and the scotoma breaks through, isolating the temporal part of the hook-shaped process, and thus producing the temporal island (Figs. 162, 163, etc.).

There is now a hemianopic scotoma with a straight edge on the vertical meridian and a rounded temporal margin extending to or enclosing the blind spot, while the remains of the temporal field, the temporal island, lie partly on and partly below the

G.P.

P

horizontal meridian beyond the blind spot. The defect now advances across the vertical meridian into the lower nasal quadrant, which fails from below upwards, while the scotoma invades its apex. The temporal island becomes gradually weaker, but can often be demonstrated until a late stage if a large enough object is used. Finally, the upper nasal quadrant becomes depressed, the scotoma extends into its apex and its last remnants usually occupy a rounded patch rather nearer the horizontal than the vertical meridian (Figs. 156, 157, 164, 165, 168, 171, 172). Thus the field changes progress regularly through the quadrants, clockwise in the right field and counter-clockwise in the left. These changes always affect both fields, though always in different degrees, so that one field presents a more advanced stage than the other. The disparity may be small, or so extreme that one field appears blind while the changes in the other may be so slight as easily to escape observation unless the examination is exhaustive. The defects are relative, becoming denser as the condition progresses, and the margins are sloping at the part where advance is occurring. As a result of this sloping edge, it is usually possible to obtain an outline showing loss of the whole temporal field alone if a suitable size of object is selected, but the true nature of the field changes can only be shown if several objects are used. The information obtained from the use of only one object is always inadequate and may even be misleading.

The progress of the defect appears to halt occasionally, the chief pause occurring when the temporal fields have practically entirely disappeared while the nasal fields are still relatively intact, so that with a test-object of moderate size a complete bitemporal hemianopia is found. A second halt may occur when the temporal field and the lower nasal quadrant have disappeared leaving a fairly clean-cut upper nasal quadrant with steep edges. Sometimes, when the progress on both sides is approximately equal, very symmetrical fields result, the two nasal halves or the two upper nasal quadrants remaining, but this is a relatively uncommon development.

The same changes take place in the colour field. Defects for colour are often found before those for white, because colour isopters, with the size of object usually employed, are internal isopters, and in depression of the field the internal isopters show alteration in position early, and, secondly, because a true disproportion may be present. It may be possible to show the progressive variation in peripheral visual acuity in all four quadrants in the typical sequence in one or both fields at the same sitting by a careful colour test. Thus red may be unrecognised in the upper temporal quadrant and only faintly in the lower, fairly well in the lower nasal and quite distinctly in the upper nasal quadrant.

The behaviour of the scotoma is very similar to that of the peripheral field. The defect commences in or near the apex of the upper temporal quadrant and may extend as far as the blind spot before it invades the apex of the lower temporal quadrant. It then affects the remaining quadrants in the same order as the peripheral changes. If when first seen it is found to occupy the apices of several quadrants, a progressive diminution in intensity in the same sequence is usually easily demonstrable by quantitative analysis. The intensity of the scotoma may be very slight in the early stages, but increases as the condition develops. In shape the scotoma as a whole may be quad-

rantic or hemianopic, or it may be round or centrocæcal, only showing its special characters on analysis. Nerve fibre bundle defects also occur, arching from the blind spot to the vertical meridian where they are sharply cut off. The fixation area is very frequently vertically divided by the edge of the scotoma, or it may be completely within the scotoma. In the latter case the vertical and horizontal meridians passing through the fixation area may still be defined by quantitative analysis, since the intensity of the scotoma varies in the different quadrants. In other cases, even in an advanced stage, when only the upper nasal quadrant remains, the fixation area is spared. This sparing may be present only for larger visual angles and absent if the test-object is small or coloured. The sparing may be greater in one field than in the other, or absent in one field, and as the case progresses the sparing usually disappears.

Non-scotomatous Type

The field changes are similar, but the scotoma is absent. In this slowly advancing type the earliest changes are best studied. It may be impossible to elicit any peripheral depression for either white or colour, the only demonstrable alterations being within the 30° circle. Here it will be found that the isopter for $\frac{1}{2000}$ does not encircle the blind spot, but leaves its upper part bare, as it were, and is deflected inwards in the upper temporal quadrant. It may or may not be possible to elicit corresponding changes for colour with certainty, as the patient may find greater difficulty in giving a definite response than when white is used (Figs. 154, 158, 159).

This early change may be in one field only, and may be inconstant, disappearing and returning again.

As the condition progresses the depression of the upper temporal quadrant becomes evident with tests such as $\frac{2}{2000}$ and $\frac{1}{330}$ white and $\frac{5}{330}$ red, and progresses gradually downwards in the temporal field. The fixation area is spared and central vision by Snellen's test remains good. The defect often does not progress into the lower nasal quadrant, and may only partially affect the lower temporal quadrant, so that only the earlier stages are attained. As a rule the same type of change is present in both fields. The non-scotomatous type may change to the scotomatous in some cases and the two types are not rigidly separated.

The changes described may be regarded as the typical or regular changes which result from interference with the chiasmal nerve path from below. Numerous variations occur both in the order in which the quadrants are affected, and in the symmetry of the two fields. The upper nasal quadrant may be affected at a relatively early stage, leading to a somewhat irregular horizontal hemianopia, or both the upper and the lower parts of the temporal field may be affected together producing a general temporal restriction, or diagonally situated quadrants may be chiefly attacked.

In an important group the defects may begin in the lower temporal quadrant, often by a scotoma in the apex of the quadrant which breaks through below in conjunction with an indentation in the lower temporal periphery, leading to an inverted uncinatè type of field.

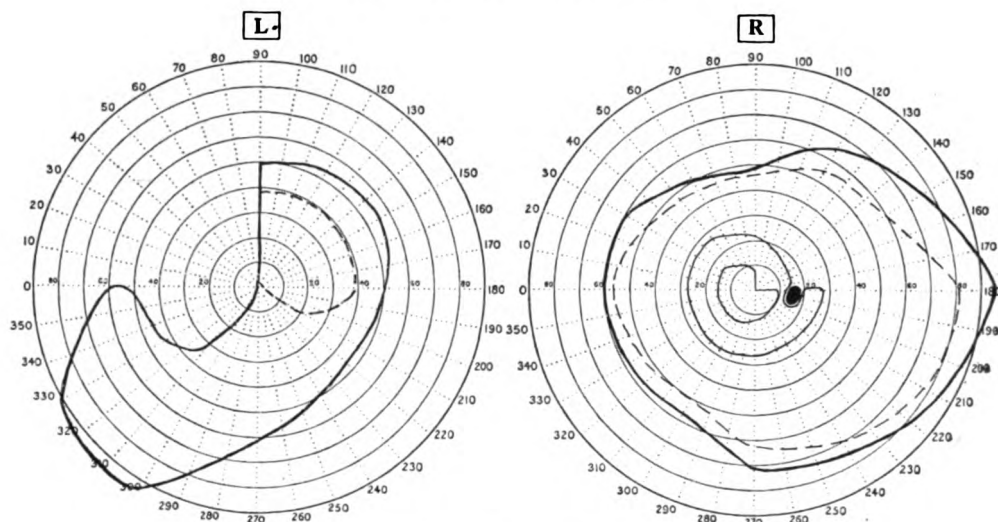


FIG. 157.—BITEMPORAL HEMIANOPIA.

Incipient changes in right field: advanced changes in left field. Left crossed fibres and some uncrossed fibres affected: right crossed fibres only very slightly involved. R.V. $\frac{5}{9}$. Objects $\frac{3}{30}$, $\frac{1}{30}$, $\frac{2}{30}$, $\frac{3}{30}$ red. L.V. $\frac{6}{24}$. Objects $\frac{3}{30}$, $\frac{1}{30}$. (H., 1930.)

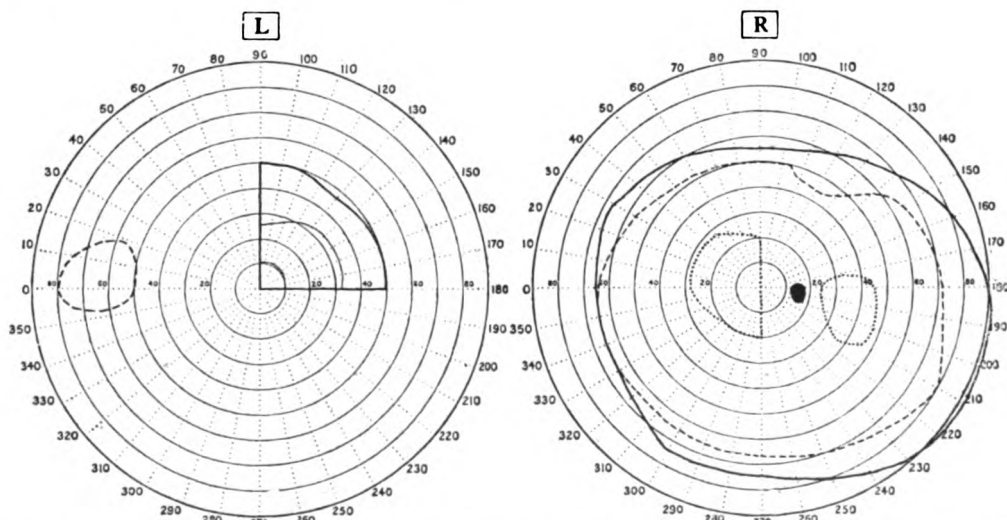


FIG. 158.—BITEMPORAL HEMIANOPIA. PENULTIMATE STAGE IN LEFT FIELD; RETENTION OF UPPER NASAL QUADRANT WITH A VERY DIM TEMPORAL ISLAND.

Objects: R. $\frac{3}{30}$, $\frac{1}{30}$: $\frac{3}{30}$ red (dotted line). L. $\frac{2}{30}$, $\frac{3}{30}$, $\frac{2}{30}$. R.V. $\frac{6}{9}$. L.V. $\frac{6}{24}$. Right field shows hemianopia for colour only and a temporal island: also a slight depression in the upper quadrant for $\frac{1}{30}$. The left field shows an upper nasal quadrant and a dim temporal island for a large object ($\frac{3}{30}$). Interference at left anterior angle of chiasma at medial side, more advanced than Fig. 156. Acromegaly absent. Headache four years, visual loss one year. (Wd., 1923.)

Not infrequently these atypical forms become modified, as the condition progresses, into more characteristic varieties, and combinations of typical and atypical forms are met with occasionally. Gradual, rapid, or sudden alterations in the distribution or character of the defects often occur, and considerable variation in central vision is not uncommon. A bitemporal hemianopia may become homonymous, the temporal half of

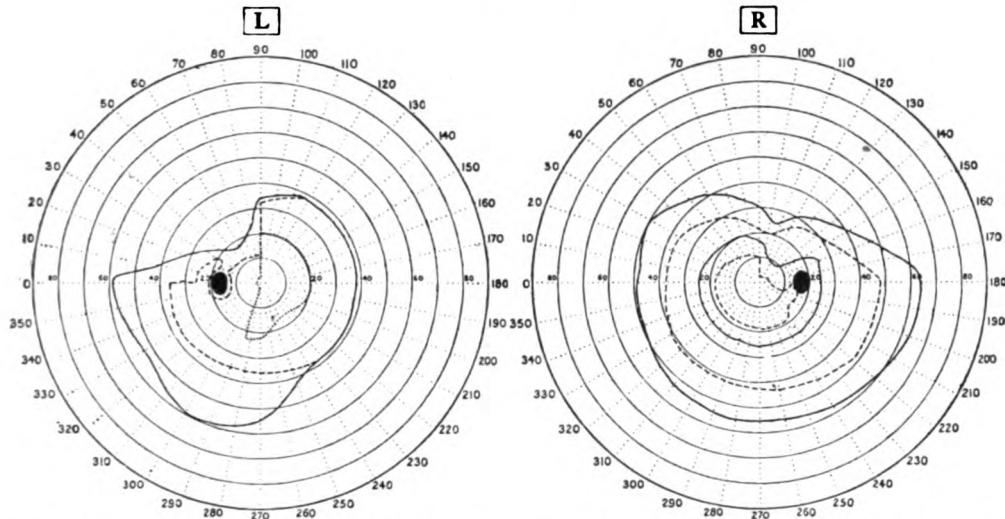


FIG. 159.—BITEMPORAL HEMIANOPIA, NONSCOTOMATOUS TYPE. LEFT FIELD SHOWS MODERATELY ADVANCED STAGE; RIGHT, EARLY CHANGES. ACROMEGALY OF FOURTEEN YEARS' DURATION.

Objects, left $\frac{3}{30}$, $\frac{1}{30}$; $\frac{1}{30}$ red (dotted line). Right, $\frac{3}{30}$, $\frac{1}{30}$; $\frac{2}{200}$, $\frac{1}{200}$. R.V. L.V. $\frac{6}{6}$. (Hh., 1912.)

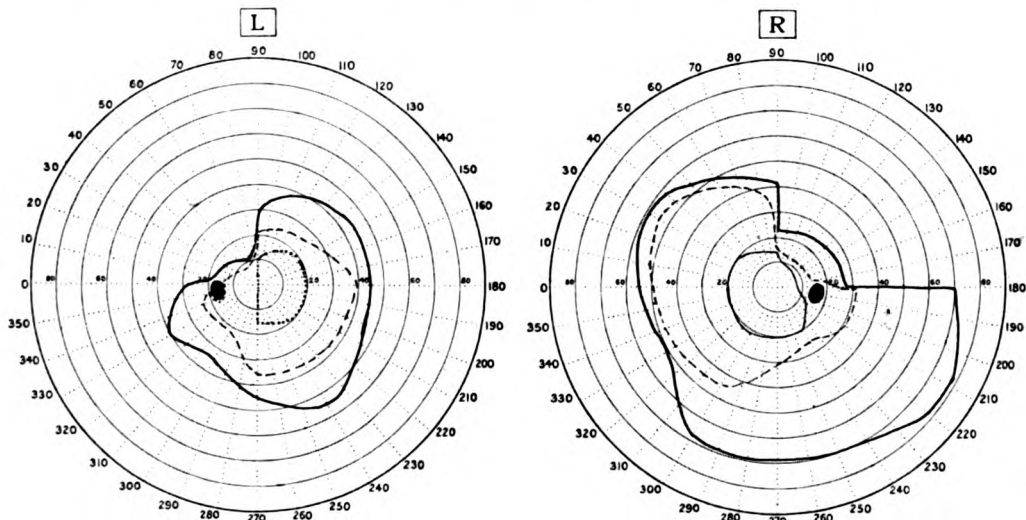


FIG. 160.—THE SAME CASE AS FIG. 159 SEVENTEEN YEARS LATER. (Hh., 1929.)

R.V. $\frac{6}{36}$. Objects: $\frac{3}{30}$, $\frac{1}{30}$, $\frac{2}{200}$.

L.V. $\frac{6}{12}$. Objects: $\frac{3}{30}$, $\frac{1}{30}$; $\frac{1}{30}$ red (dotted line).

Note the almost stationary condition of the fields over a long period. The fall in vision is due to retinal disease.

one field recovering while the nasal half becomes affected, a change sometimes associated with a halt in the progress of the defects and an improvement in vision in one or both eyes. Practically any combination of stages or forms may be present at one time in the two fields, and the difference between the fields may be extreme. Total blindness on one side with a hemianopic or quadrantic defect in a very early stage in the opposite field may be found and may easily be misinterpreted, or the slight defect may escape observation. In the later stages the typical development may be greatly modified.

and the ultimate picture may be atypical in one or both fields. A final remnant may be present in one lower nasal quadrant, and symmetrical retention of both lower nasal quadrants, a great rarity, has been recorded by Hirsch (182), and, in a case of injury, by Liebrecht (247).

When recovery occurs, as for example, following a successful operation, the field changes regress in the same way as they advanced. The process may stop at any stage according to the amount of irreparable damage done. When the field finally appears

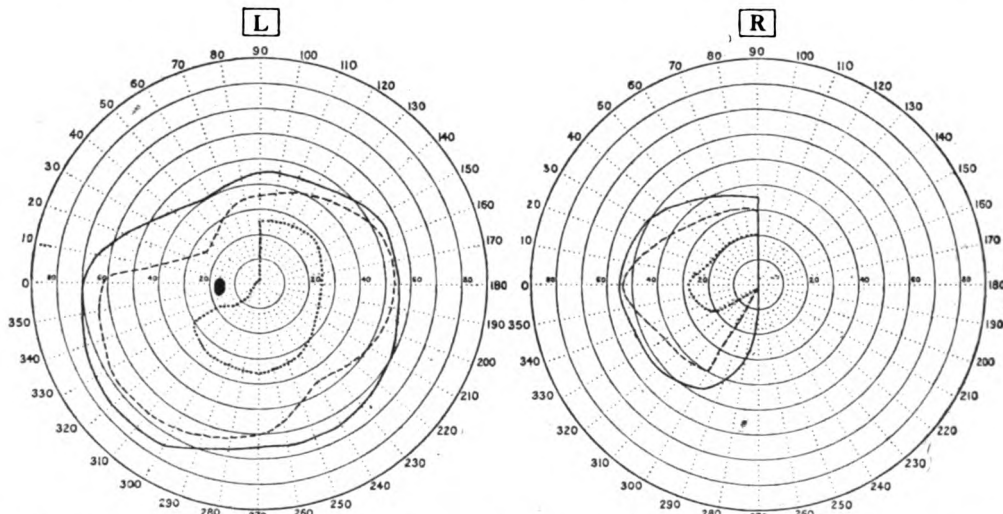


FIG. 161.—BITEMPORAL HEMIANOPIA. EARLY STAGE ON LEFT SIDE WITH SPARING OF FIXATION AREA; COMPLETE TEMPORAL HEMIANOPIA ON RIGHT SIDE WITH DIVIDED FIXATION AREA.

R.V. $\frac{6}{30}$. L.V. $\frac{6}{24}$. Gland symptoms minimal.

Objects: R. $\frac{3}{30}$, $\frac{1}{30}$: red $\frac{5}{30}$ (dotted line). L. $\frac{3}{30}$, $\frac{1}{30}$: red $\frac{1}{30}$ (dotted line). (Pe., 1924.)

to be quite normal it is often possible to detect a slight inward deflection of the $\frac{1}{2000}$ isopter in the upper temporal quadrant exactly similar to the signs found in the early stage of advance.

It will be evident from the manner in which these field changes develop that they cannot be detected in the early stages, and will not be adequately demonstrated at any stage, unless more than one size of test-object is used including, especially in incipient cases, a very small object such as $\frac{1}{2000}$ or a small coloured object.

Interpretation.—The study of chiasmal field defects affords the best introduction to the principle of anatomical interpretation based on the fascicular architecture of the visual path. In the optic nerves the fibres from contiguous parts of each retina lie together, and bilateral field defects are the expression of separate lesions and have individual and independent features. At the chiasma the crossed and uncrossed fibres become separated, then intermingled, and finally regrouped into the tracts. The widespread arrangement of the crossing fibres flanked by the direct bundles makes the chiasma the only place where a single point of interference can produce bitemporal

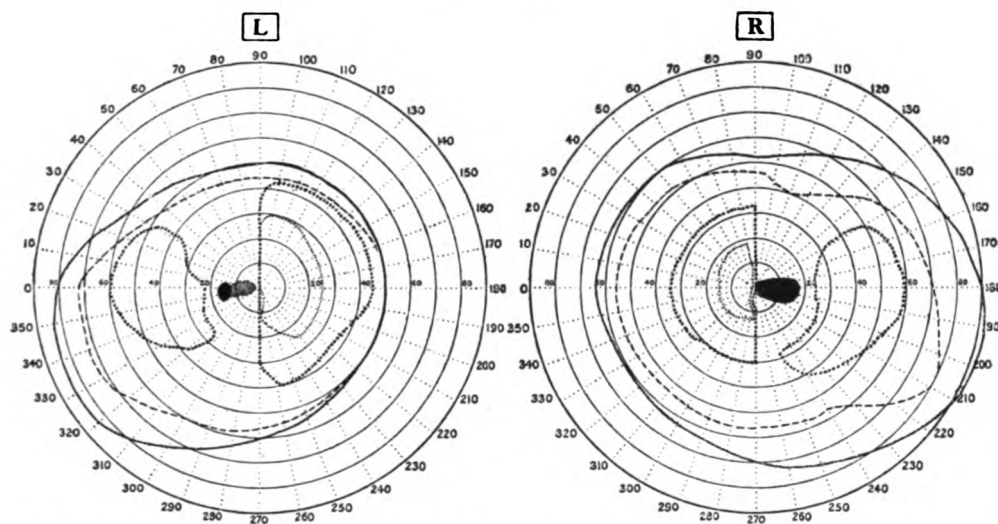


FIG. 162.—BITEMPORAL HEMIANOPIA. WELL-DEVELOPED CHANGES, ESPECIALLY FOR COLOUR. CENTROCÆCAL SCOTOMA, SPLITTING OFF OF TEMPORAL ISLAND, COMPLETE HEMIANOPIA FOR SUITABLE OBJECT.

Objects : $\frac{5}{330}$, $\frac{3}{330}$; $\frac{20}{330}$, $\frac{10}{330}$ red. Acromegaly : four years after onset of gland symptoms. R.V. $\frac{6}{60}$. L.V. $\frac{6}{12}$. (Wn., 5/4/12.)

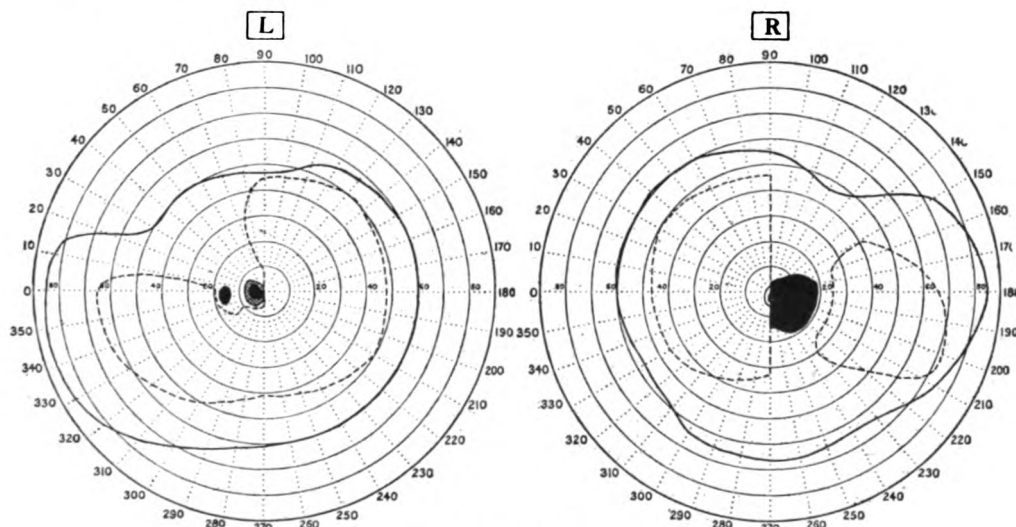


FIG. 163.—THE SAME CASE AS FIG. 162 ELEVEN WEEKS LATER.

Pronounced changes for white now present. On the right side the scotoma is invading the nasal field. R.V. $\frac{3}{60}$. L.V. $\frac{6}{12}$. (Wn., 21/6/12.)

hemianopia, and the complicated fascicular crossings enable a single lesion at different parts to produce various combinations of field defects. For the same reason it is unlikely that even a minute lesion, if in the chiasmal body, will cause a defect limited to one field. A median lesion involves both crossed bundles, and a lateral lesion both crossed and one direct bundle. Nor can the macular fibres be easily affected alone, so that bitemporal hemianopic central scotomata are almost always accompanied by some surrounding defect. The fibres from each retina preserve their original relationships at this level,

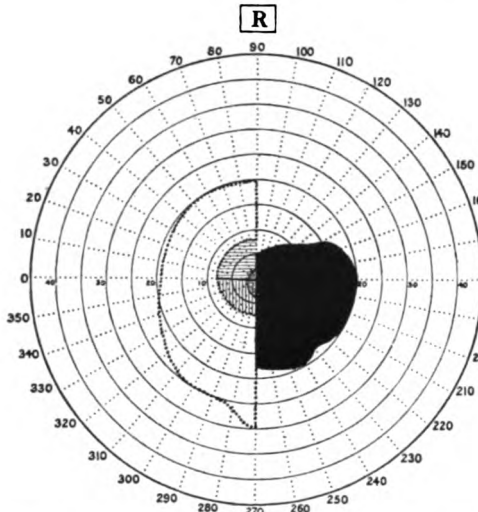


FIG. 164.—ANALYSIS OF THE SCOTOMA IN THE RIGHT FIELD OF FIG. 163.

Absolute scotoma for $\frac{30}{2000}$ white, too dense to analyse. Small nasal defect for $\frac{1}{2000}$ white, larger in lower quadrant. Hemianopic central defect for $\frac{10}{330}$ red (dotted lines) in which red was called orange in upper part (horizontal shading) and yellow in lower part (vertical shading). (Wn., 5/6/12.)

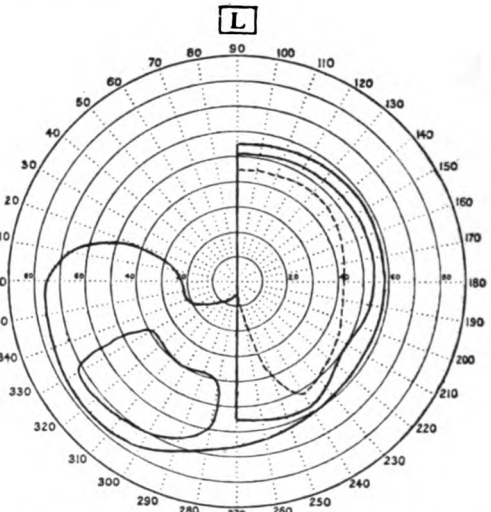


FIG. 165.—THE LEFT FIELD OF THE SAME CASE TWO MONTHS LATER, SHOWING TYPICAL CHANGES.

Objects $\frac{40}{330}$, $\frac{10}{330}$, $\frac{1}{330}$. V. = $\frac{5}{10}$. The right eye was almost completely blind at this time. (Wn., 19/8/12.)

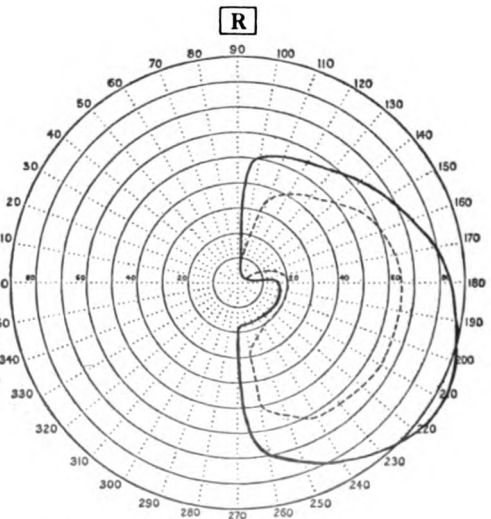
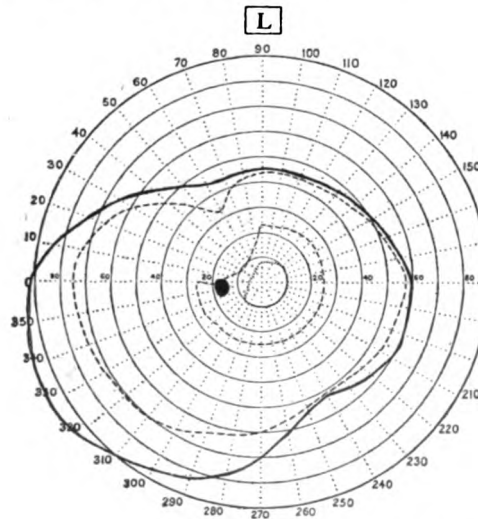


FIG. 166.—THE SAME CASE TEN YEARS LATER.

Almost complete recovery of left field which shows only the earliest stage. Objects $\frac{330}{330}$, $\frac{330}{330}$, $\frac{2000}{330}$; $\frac{1000}{330}$ red. V. = $\frac{6}{6}$. The right field shows nasal hemianopia with central defect. Objects $\frac{330}{330}$, $\frac{330}{330}$. V. = Hand movements. The change to homonymous hemianopia occurred two months after the date of Fig. 163 by the reappearance of the right temporal and complete loss of the right nasal field. (Wn., 28/8/22.)

excepting in so far as the crossed and uncrossed bundles are separated, and those from corresponding points in the two retinae have not yet come to lie side by side. Thus the characteristic features of chiasmal field defects depend on interference with more than

one fasciculus, which can hardly be avoided even by a very small lesion owing to the intermingling of the fibres, and the defects vary in extent and intensity in the two fields according to the way in which more or less of the fibres of the bundles concerned are affected.

Bitemporal hemianopia indicates an interference with the crossed fibres from both retinae, and, therefore, a lesion in or near the median plane affecting either the medial sides of both optic nerves, the body of the chiasma, or possibly the medial sides of both tracts close behind the chiasma. A tumour growing between the nerves usually affects one before the other and produces a unilateral temporal hemianopic defect which may exist for some time before the other field is involved. Then as the crossed fibres of the other nerve become involved, slight temporal hemianopia appears in the other field and the

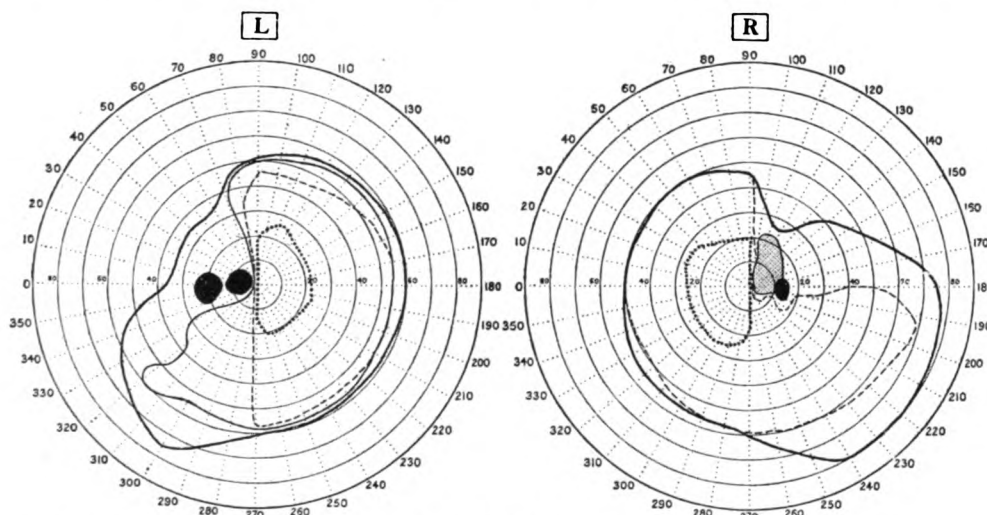


FIG. 167.—BITEMPORAL HEMIANOPIA. TYPICAL FIELDS IN A MODERATELY ADVANCED CASE. Hypophysial tumour without acromegaly. Duration one year. Objects $\frac{1}{2} \frac{5}{10}$, $\frac{2}{2} \frac{5}{10}$, $\frac{1}{2} \frac{5}{10}$; $\frac{2}{2} \frac{5}{10}$ red. R.V. $\frac{6}{6}$. L.V. $\frac{6}{6}$. (J., 11/8/15.)

original defect progresses to blindness leading to a typical condition :—blindness of one eye with temporal hemianopia in the other. The third possibility is more theoretical than actual as a tumour or other lesion between the tracts would require to exert its influence towards both sides but not forwards in order to produce bitemporal hemianopia from tract interference alone. Whether this ever occurs is not known, and does not seem probable ; tumours in this position usually cause homonymous hemianopia passing on to blindness of the eye on the side opposite to the originally affected half-fields, since complete blockage of one tract is followed by interference with the crossed fibres of the other and the chiasma becomes involved.

As a rule a median tumour presses upwards beneath the chiasma and between the nerves, then as it enlarges further the main growth is between the tracts, tipping the chiasma forwards. The chiasma may be stretched over the surface of the tumour or may be canted into an almost vertical position. At first fibres from the middle and apical parts of the lower medial retinal quadrants are attacked. On anatomical grounds

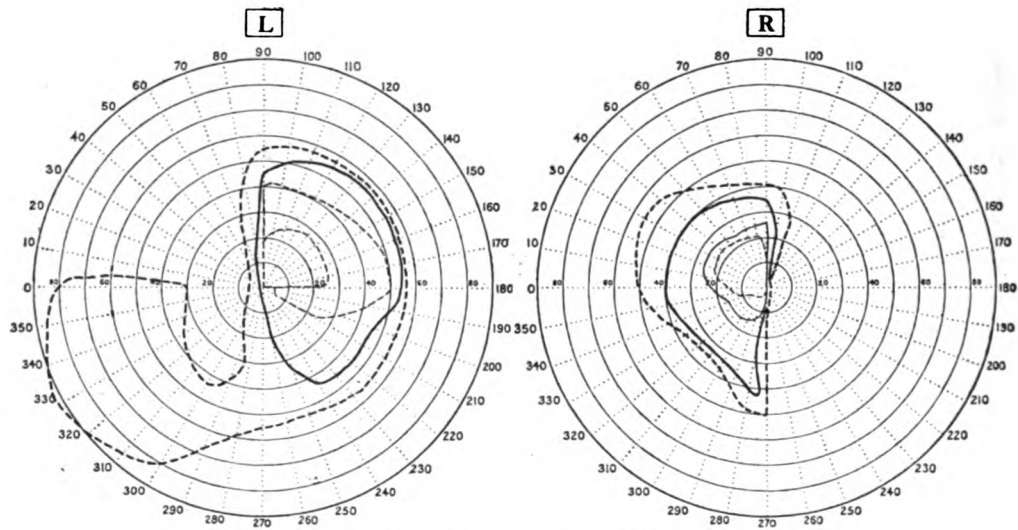


FIG. 168. BITEMPORAL HEMIANOPIA. TERMINAL STAGES BEGINNING.

The lower nasal fields are gradually shrinking upwards. Temporal field partly present on left side for a large object. Acromegaly.

Objects: R. $\frac{4}{30}$, $\frac{5}{30}$, $\frac{2}{60}$, $\frac{1}{60}$. V. $\frac{6}{30}$.

L. $\frac{4}{30}$, $\frac{5}{30}$, $\frac{2}{60}$, $\frac{1}{60}$. V. $\frac{6}{30}$.

Same case as Fig. 154 three years later. (B., 15/3/16.)

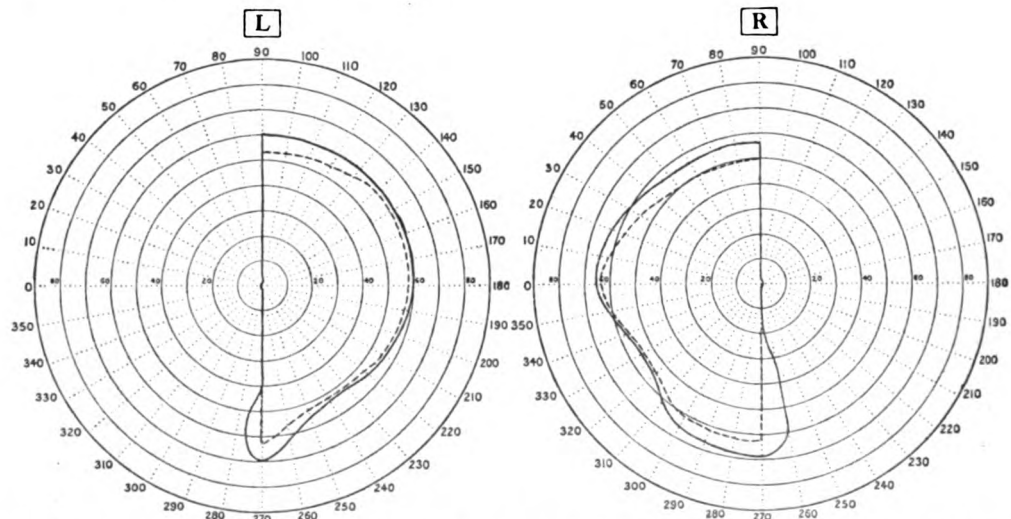


FIG. 169.—BITEMPORAL HEMIANOPIA. A CLOSE APPROACH TO COMPLETE LOSS OF TEMPORAL FIELDS WITH INTACT NASAL FIELDS. SPARING OF FIXATION AREA.

Objects, $\frac{5}{30}$, $\frac{1}{30}$. Some very dim vision for large objects still present in temporal fields. R.V. $\frac{6}{30}$. L.V. $\frac{6}{30}$.

Same case as Fig. 167 five years later. Patient died two years later without notable further change in the fields. (J., 11/8/20.)

it is clear that any median interference from below will involve both sets of crossed fibres, and the more the posterior edge of the chiasma is affected the more prominent will be the scotomatous character of the field changes. The fibres from the upper medial retinal quadrants come next in order, presumably because they lie superiorly in the chiasma. The sparing of the temporal island is a very characteristic feature in tumour

cases and must have some significance. It seems justifiable to assume that the fibres which are represented by the temporal island do not lie among the crossing fibres which traverse the centre of the chiasma, but probably more laterally in the neighbourhood of, or mingled with, the direct fibres. The presence of the temporal island with comparatively intact nasal fields indicates that the site of interference is approximately median, for were the crossing fibres blocked where they are laterally placed, the direct fibres would suffer also. Ultimately when the chiasma becomes practically divided antero-posteriorly, the island disappears, and by this time the superior direct fibres are

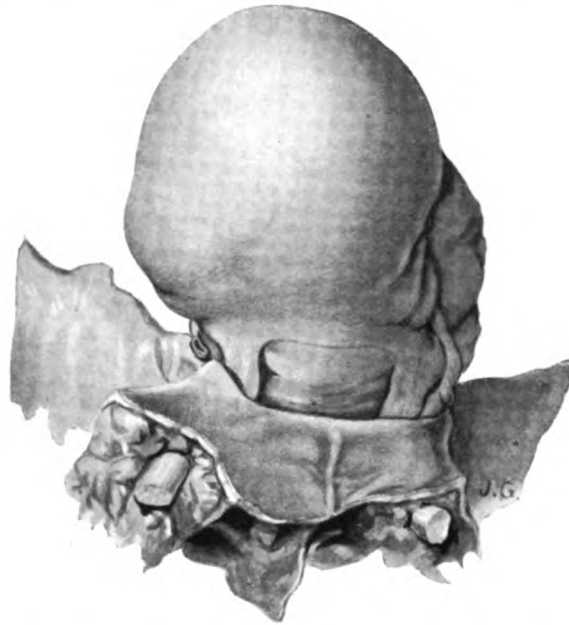


FIG. 170.—TUMOUR FROM SAME CASE AS FIGS. 167, 169.

To illustrate antero-posterior splitting of chiasma by a large centrally placed tumour whose continued upward growth does not further affect the nasal fields. The tumour is seen from in front with the remains of the chiasmal body, slightly exaggerated in the drawing, stretched across its anterior surface. Partially degenerated nerve fibres were found crossing the front of the tumour. (Natural size.)

showing signs of impaired function. The chiasma is now much distorted, the crossed fibres are apparently entirely obliterated, and the uncrossed bundles lie at the sides of the tumour comparatively unharmed.

The temporal island is found in bitemporal hemianopia from various causes, and its presence appears to indicate the chiasma as the site of interference. It is apparently absent when the lesion is prechiasmal and affects the medial sides of the two nerves, presumably because the crossed bundle is not widely spread out in the nerve as it is in the chiasma, but in the present state of our knowledge it would be unwise to interpret its absence always in this way.

At this stage, when the temporal fields are practically blind and the nasal fields not yet seriously affected, a halt is apt to occur, and it might be supposed that the tumour had ceased to grow. Good central vision may be retained on one side if not

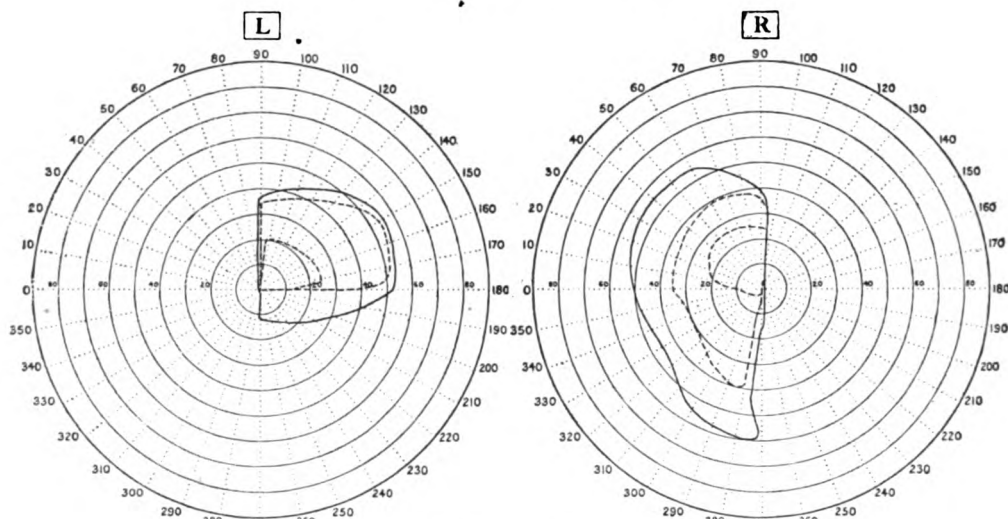


FIG. 171.—BITEMPORAL HEMIANOPIA. LATE STAGES.

Same case as Fig. 155 four years later, one year later than Fig. 168. Continued shrinkage of left field, some improvement in right. Objects $\frac{3}{30}$, $\frac{1}{30}$; $\frac{1}{2000}$. R.V. $\frac{6}{18}$. L.V. $\frac{6}{36}$. (B., 23/5/17.)

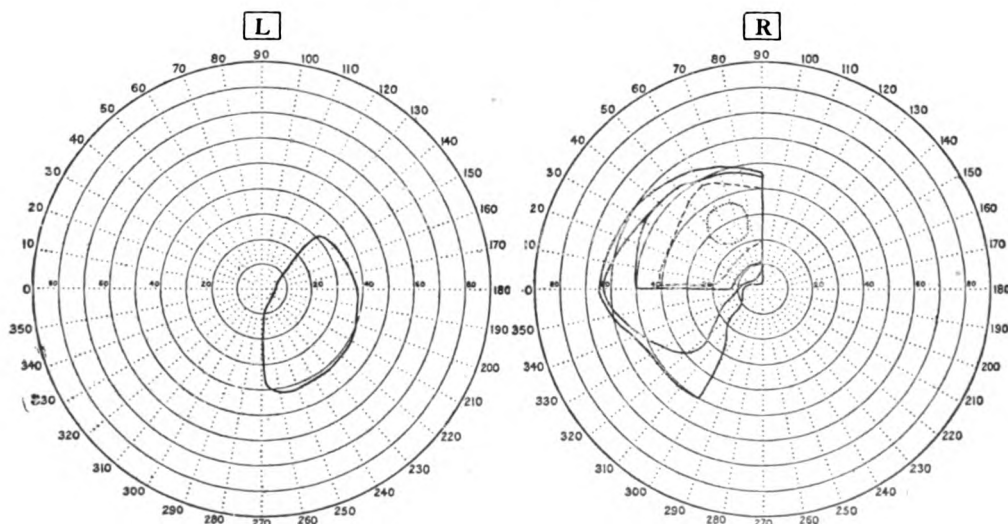


FIG. 172.—BITEMPORAL HEMIANOPIA. LATE STAGES. ATYPICAL TERMINATION IN ONE FIELD.

Very dim lower nasal quadrant in left field. Object $\frac{6}{30}$. V. = hand movements.

Typical late stages in right field; objects $\frac{6}{30}$, $\frac{3}{30}$, $\frac{3}{30}$, $\frac{3}{30}$; $\frac{3}{30}$ red (dotted line). V. = counting fingers at 1 m. Four years after commencement of glandular symptoms (mixed type). (Hg., 1924.)

on both, and the nasal fields remain relatively intact, but the other symptoms, such as headache, persist. What actually happens is that the tumour, having split the chiasma antero-posteriorly, grows up into the brain. The uncrossed bundles continue along the sides of the tumour comparatively unharmed into the tracts, and the crossed fibres are non-existent, though their remnants may sometimes be traced across the front of the tumour. Death may occur with the fields still in this state.

The failure of the temporal field from above downwards is not difficult to under-

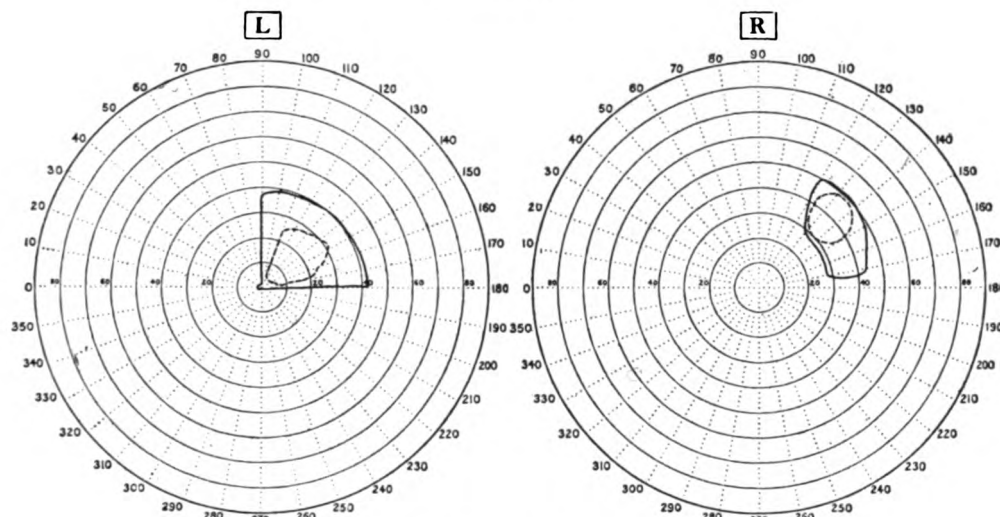


FIG. 173.—BITEMPORAL HEMIANOPIA TERMINATING HOMONYMOUSLY.

Upper nasal quadrant remaining in left field; objects $\frac{3}{3} \frac{0}{0}$, $\frac{2}{0} \frac{0}{0}$. V. = Fingers at 4 m. Upper temporal quadrant remnant in right field: objects $\frac{6}{3} \frac{0}{0}$, $\frac{3}{3} \frac{0}{0}$. V. = perception of hand movements. Eighteen months after onset, acromegaly not present. (W. S. B., 1922.)

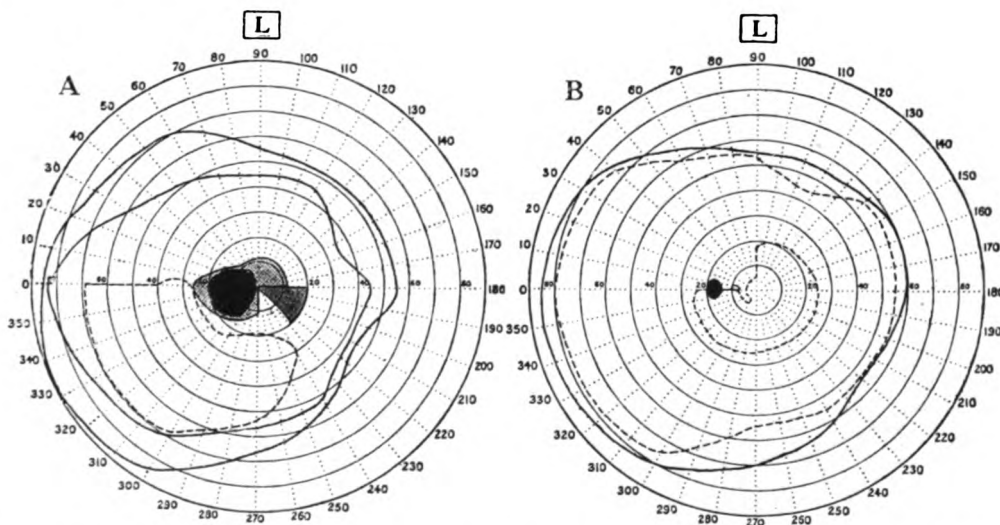


FIG. 174.—BITEMPORAL HEMIANOPIA. ILLUSTRATING SPONTANEOUS RECOVERY IN HYPOPHYSEAL TUMOUR.

A. Field of L.E. showing depression and central defects. Periphery for $\frac{2}{3} \frac{0}{0}$. Retained area in lower temporal quadrant for $\frac{3}{3} \frac{0}{0}$. Central scotomatous area too defective to be easily analysed. V. = $\frac{1}{\pi} \frac{0}{0}$.
 B. Three months later V. $\frac{2}{3} \frac{0}{0}$. Field normal for $\frac{2}{3} \frac{0}{0}$, and $\frac{1}{3} \frac{0}{0}$, quadrant defect for $\frac{3}{0} \frac{0}{0}$ and small scotoma for $\frac{3}{0} \frac{0}{0}$. The field of R.E. showed a typical temporal hemianopia and recovered also very considerably, but not so much as L. Eighteen months later both fields were very defective and the patient ultimately became blind. Same case as Fig. 172. (Hg., 1920.)

stand in the case of a lesion advancing from below. The explanation of the upward failure of the nasal field is not so evident. Possibly the superior uncrossed fibres fail first because they lie more centrally in the chiasma, or possibly they are not affected in the chiasma, but farther forwards on account of pressure on the upper surfaces of

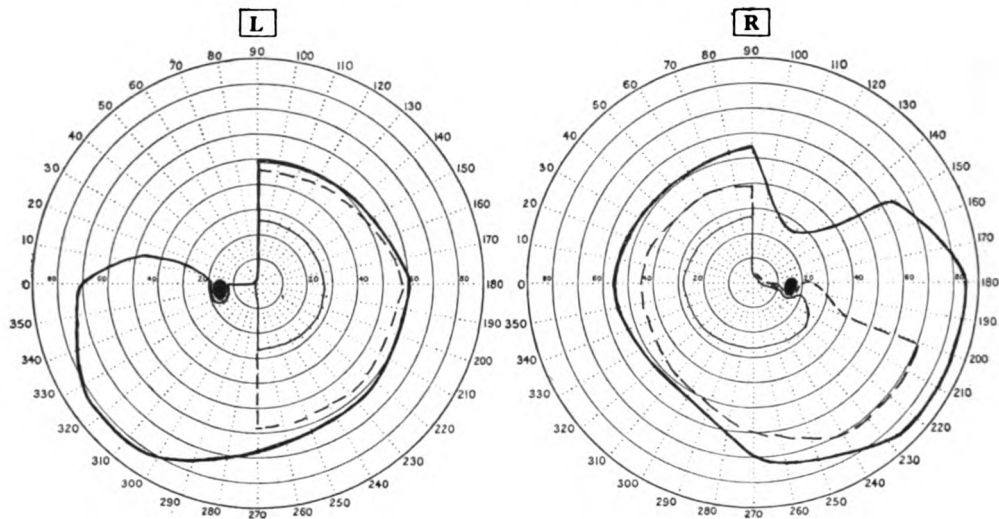


FIG. 175.—BITEMPORAL HEMIANOPIA. BEFORE OPERATION.

R.V. 5. L.V. 6. Objects $330, 340, 2000$. Pronounced upper quadrant defects. (S., 1933)

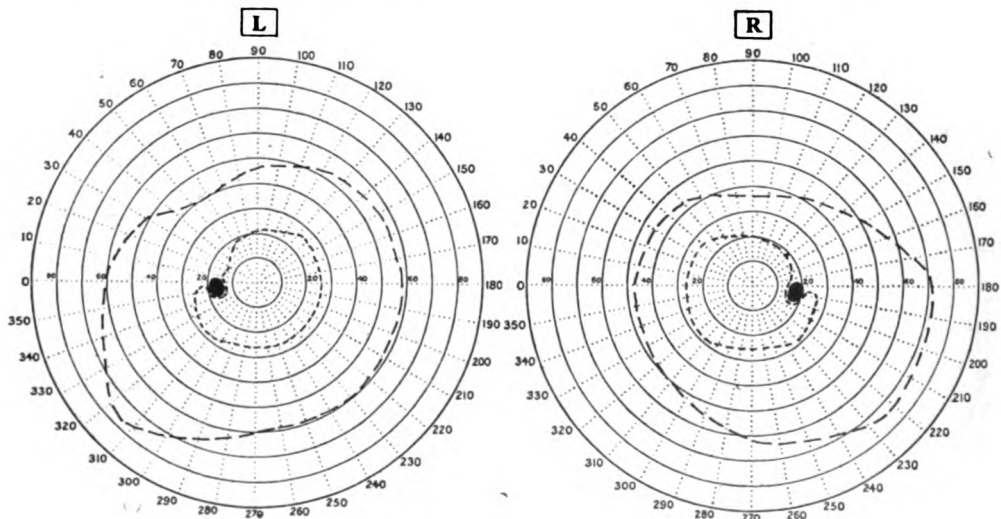


FIG. 176.—POST-OPERATIVE RECOVERY. SAME CASE AS FIG. 175.

R.V. 4. L.V. 4. Objects $330, 2000$. Only traces of upper temporal quadrant depression remain. (S., 1933.) Compare with Fig. 154.

the nerves by the vessels of the anterior arch of the circulus arteriosus * (Lillie 253), or by the fold of dura covering the intracranial end of the optic foramen. There is considerable evidence, however (McConnell and Mooney 270, Jefferson 207), that the affection of the lower nasal quadrants occurs independently of indentation of the upper surfaces of the optic nerves. The causal lesion need not be a tumour and there may be no indentation of the nerves. The presence of the peculiar sequence of the field defects at an early

* But see Hirsch (182), Case 7. Here the upper surfaces of the nerves were deeply indented by the arteries, but the lower nasal quadrants were preserved.

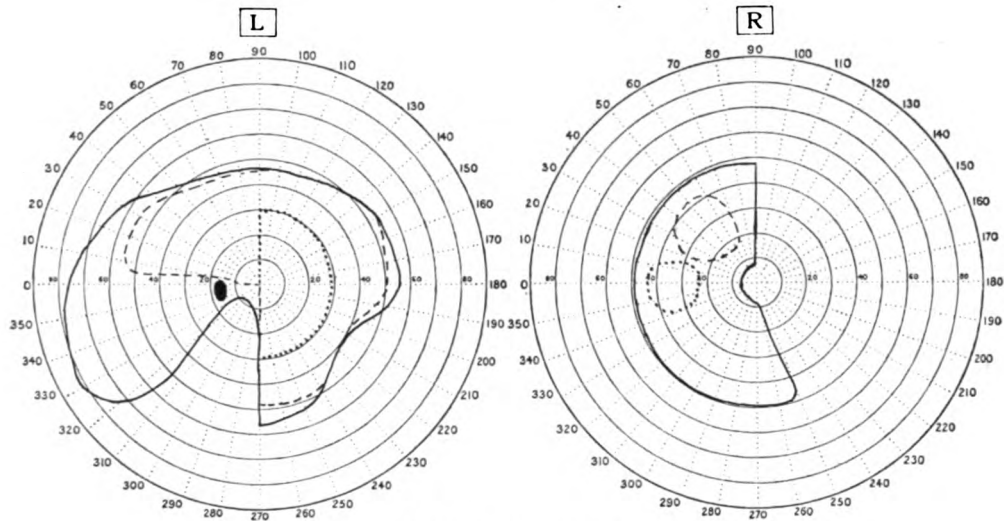


FIG. 177.—BITEMPORAL HEMIANOPIA FROM SUPRASELLAR CYST.

The right field was too defective to chart accurately: the field for a large object was approximately as shown, the best vision being retained in the upper nasal quadrant. V. H.M. Red = dotted line: $\frac{3}{30}$ = broken line. In the left field there was complete temporal hemianopia for $\frac{10}{30}$ red with a lower temporal quadrant defect for $\frac{1}{30}$ white. For a larger object ($\frac{3}{30}$) a wedge-shaped defect was present in the same quadrant. Objects: $\frac{1}{30}$ red. (A. S., 1927.)

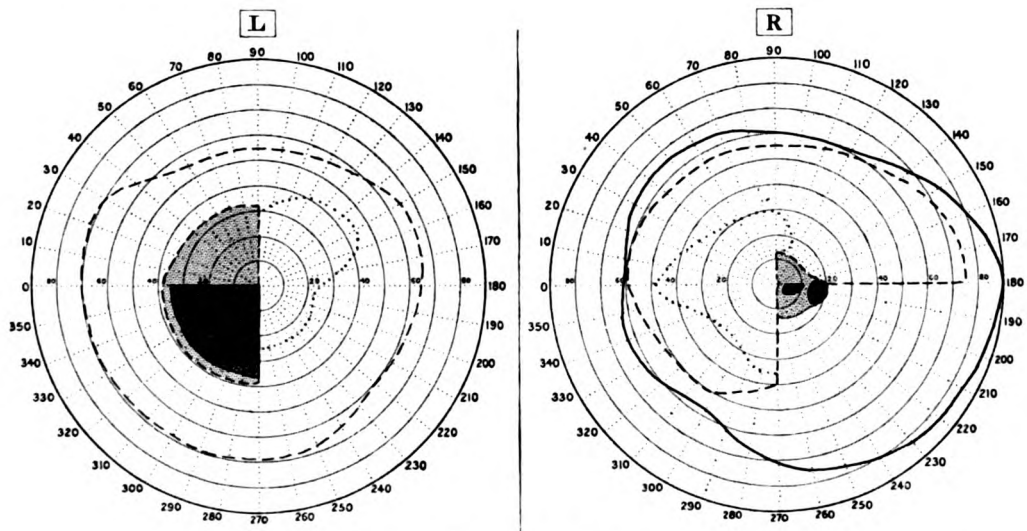


FIG. 178.—BITEMPORAL HEMIANOPIA.

Defect more severe in lower temporal quadrants. From a case of suprasellar cyst. R. V. $\frac{6}{9}$. L.V. $\frac{6}{18}$. Objects: $\frac{3}{30}$ red, $\frac{10}{30}$ red (nasal fields only). The hemianopic scotomata were defective for $\frac{3}{30}$ and $\frac{6}{30}$ (darker areas). Field for $\frac{3}{30}$ in left eye omitted. (G., 1926.)

stage, whether the lesion is a tumour or not, suggests that these uncrossed fibres are affected in the chiasma and that the sparing of the lower uncrossed fibres until the last stage is to be attributed to their blood-vessels being sheltered in some way so that normal circulation can be maintained amongst them longer than in the upper group. It may be justifiable to infer that in the chiasmal body the lower uncrossed fibres lie

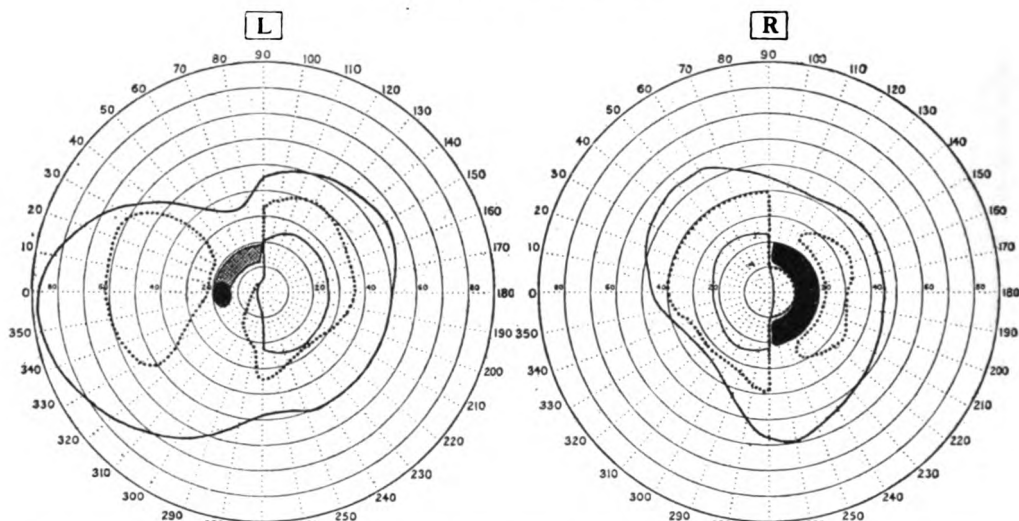


FIG. 179.—BITEMPORAL HEMIANOPIA. ATYPICAL CONDITIONS. NERVE FIBRE BUNDLE DEFECTS.
Objects $\frac{3}{30}$, $\frac{3}{30}$: $\frac{10}{30}$ red (dotted line). R.V. = $\frac{6}{6}$ —. L.V. $\frac{6}{6}$ —, acromegaly. Same case as Fig. 155. (B., 1914.)

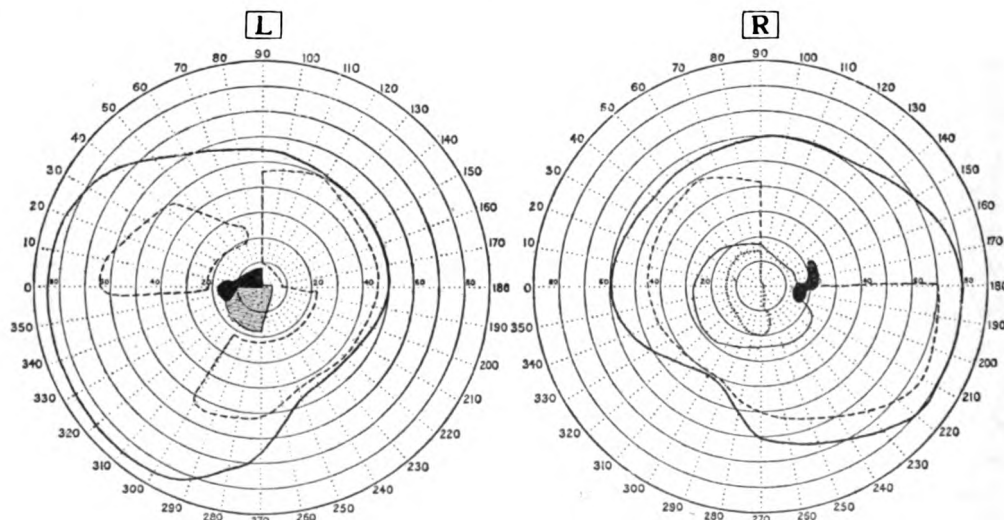


FIG. 180.—BITEMPORAL HEMIANOPIA. IRREGULAR CHANGES ON LEFT SIDE, REGULAR ON RIGHT WITH NERVE FIBRE BUNDLE DEFECT.

Objects R. $\frac{3}{30}$, $\frac{3}{30}$, $\frac{20}{50}$: $\frac{10}{50}$ red (dotted line). L. $\frac{3}{30}$, $\frac{3}{30}$. Scotoma, $\frac{20}{50}$, $\frac{20}{50}$. R.V. $\frac{6}{6}$. L.V. $\frac{6}{6}$. Same case as Fig. 172 four years previously. (Hg., 6/5/20.)

laterally in relation to the upper uncrossed fibres, a surmise which, if established, might explain some of the atypical cases.

The mechanism of production of the field defects has been the subject of three hypotheses: pressure, traction, and the action of toxins. Of these by far the most attractive is the first, provided that it is understood that the pressure acts not directly on the nerve fibres but on the blood-vessels, by impeding the venous return and by producing arterial ischæmia. Purves Stewart and Riddoch have shown that this

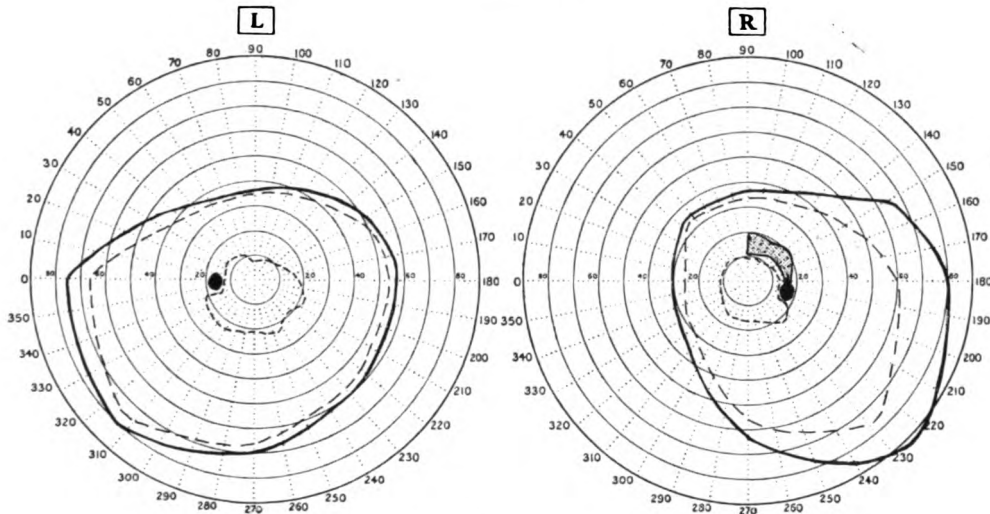


FIG. 181.—BITEMPORAL HEMIANOPIA TWO YEARS AFTER OPERATION.

Right field shows a nerve fibre bundle defect in upper temporal quadrant. Left field shows remains of upper temporal quadrant defect.

R.V. $\frac{9}{8}$. Objects $\frac{3}{30}$, $\frac{3}{30}$, $\frac{4}{30}$ blind spot, $\frac{2}{30}$ scotoma, $\frac{1}{30}$.

L.V. $\frac{9}{8}$. Objects $\frac{3}{30}$, $\frac{3}{30}$, $\frac{4}{30}$, $\frac{1}{30}$. (S., 1934.)

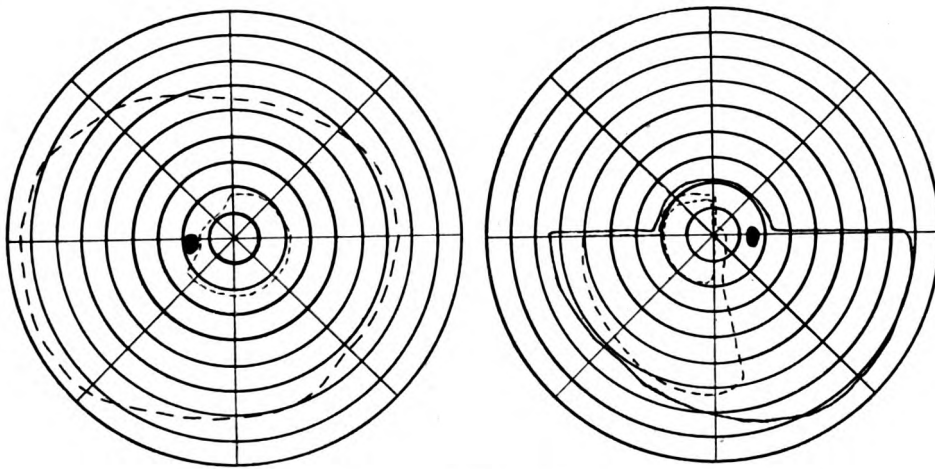


FIG. 182.—BITEMPORAL HEMIANOPIA FROM INTRASSELLAR TUMOUR.

Atypical changes in the right field: typical early changes in the left field. Showing the necessity of examining the apparently normal field. R.V. $\frac{9}{8}$. L.V. $\frac{9}{8}$. (A., 1942.)

process is the essential factor in pressure on the spinal cord, and it is difficult to explain the features of chiasmal pressure interference on any other basis. Such a process could enable a rounded surface like that of a tumour to give rise to field defects with sloping edges which recover rapidly when the pressure is relieved. On this hypothesis the production of clean-cut bitemporal hemianopic fields without the necessity of invoking a "knife-edge" form of pressure can be explained, and also the resemblance between the effects of pressure, inflammation, and traumatism.

C.P.

9

The significance of the scotoma is of interest. It is present in inflammatory cases and in tumour cases when the growth is more or less active; it is absent when the tumour is of slowly growing or practically stationary type as in chronic or "benign" acromegaly. It is possible that it is the activity of growth rather than the site of the interference which determines the presence of the scotoma, but at the same time we must remember that the more rapidly growing tumours may originate in the hypophysial stalk or, if intrasellar, enlarge the sellar orifice above, and press up against the back of the chiasma where the central fibres cross. According to Jefferson (207) bilateral scotomatous fields suggest prefixation of the chiasma and the presence of a large tumour. Inflammatory processes such as gummatous meningitis also tend to involve the back of the chiasma. When the tumour is intrasellar and slowly growing the dorsum sellæ is not eroded but thinned and expanded, and appears elongated upwards, and the

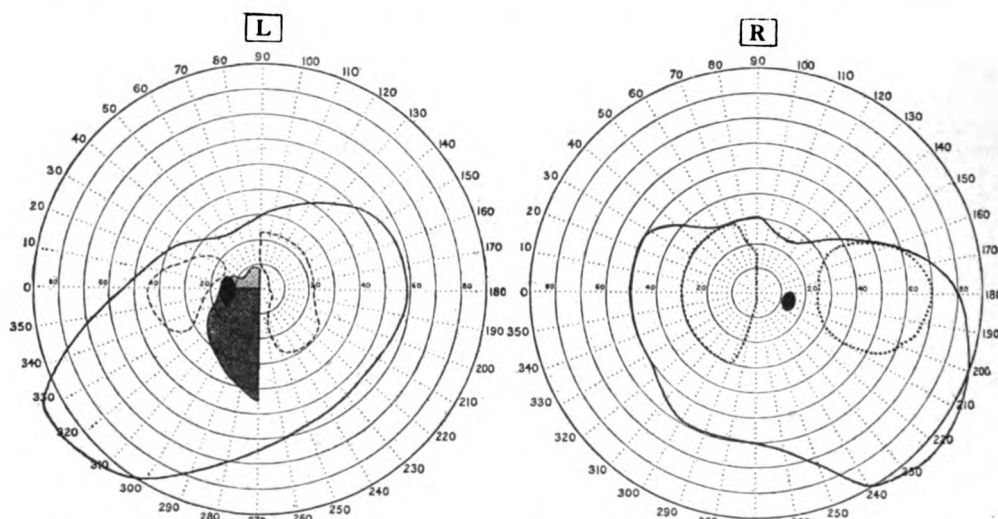


FIG. 183.—BITEMPORAL HEMIANOPIA. IRREGULAR CHANGES IN LEFT FIELD, REGULAR IN RIGHT.

Objects : $\frac{2}{50}$: $\frac{10}{250}$ red (dotted line). R.V. $\frac{9}{9}$: L.V. nearly $\frac{6}{6}$. Acromegaly not present. Duration of symptoms six months. (Wnn., 7/7/1915.)

tumour enlarges more in a forward direction. Many intermediate and varying modes of growth occur, and, no doubt, account for the different degrees of prominence of the scotoma in individual cases and at different times in the same case.

Diagnosis.—In the diagnosis of the presence and cause of interference with the chiasma in relation to morbid processes in its neighbourhood, the assistance afforded by perimetry is of value chiefly in two groups of cases. Firstly, those in which visual loss attracts the attention of the patient before other symptoms emerge. Such patients apply to the ophthalmic surgeon and exhibit well-marked field changes, especially centrally, which frequently provide the first evidence of the true nature of the condition. The perimetric evidence is of greater importance in those cases in which the radiographic examination is negative, and often provides the only indication of the nature of the disease. Many of these cases are misdiagnosed or inadequately diagnosed and the patients are subjected to unnecessary exploration of the nasal sinuses, extraction of

teeth, and other measures, when a thorough field examination would have indicated the true solution. Secondly, those in which symptoms suggestive of altered endocrine secretion or intracranial lesion have been found by the physician, and it is desired to know whether, and in what way, the visual path is affected. In these cases early field defects unnoticed by the patient may be found. Since the effect on the visual path is produced by actual contact, cases of hypophysial enlargement do not show visual symptoms before they show sellar changes, though suprasellar growths produce visual defects at an early stage before radiographic changes are evident. The perimetric evidence here supplements the radiograph and gives a hint as to the extent to which the swelling is elevated above the normal level of the diaphragma sellæ.

Pallor of the optic discs usually appears slowly and in connection with well-developed field changes. When these are very slight it may remain absent.

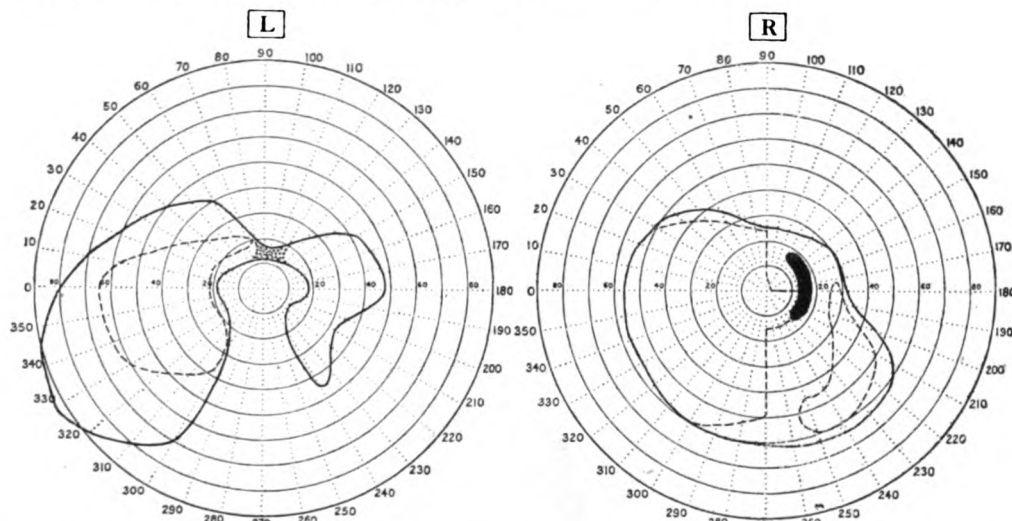


FIG. 184.—BITEMPORAL HEMIANOPIA. SAME CASE AS FIG. 183 THREE MONTHS LATER. CHANGING TO HOMONYMOUS DEFECT.

Objects, $\frac{5}{200}$, $\frac{1}{15}$. R.V. = $\frac{6}{18}$. L.V. = hand movements. The isthmus in left field is very dim for $\frac{5}{250}$. (Wnn., 18/10/15.)

For the clinician various questions arise :—

I. The localisation of the interference.

- (a) Localisation of the interference at the chiasma.
- (b) The part of the chiasma affected.

II. The cause of the interference.

- (a) Tumour. Size, rate of growth, nature.
- (b) Causes other than tumour.

We may now examine the extent to which perimetric findings can help towards the solution of these problems.

I. The Site of the Interference

(a) Localisation at the chiasma.

If changes are found which suggest a temporal contraction care must be taken to

ascertain its true nature. It has already been pointed out that uniform depression of the field produces an apparent temporal restriction owing to the less steep slope of the temporal side. A field change of this kind shows no evidence of quadrant depression, and if thoroughly examined will show some concentric restriction of the nasal isopters also. In suspected cases, where there is no scotoma and the peripheral changes are

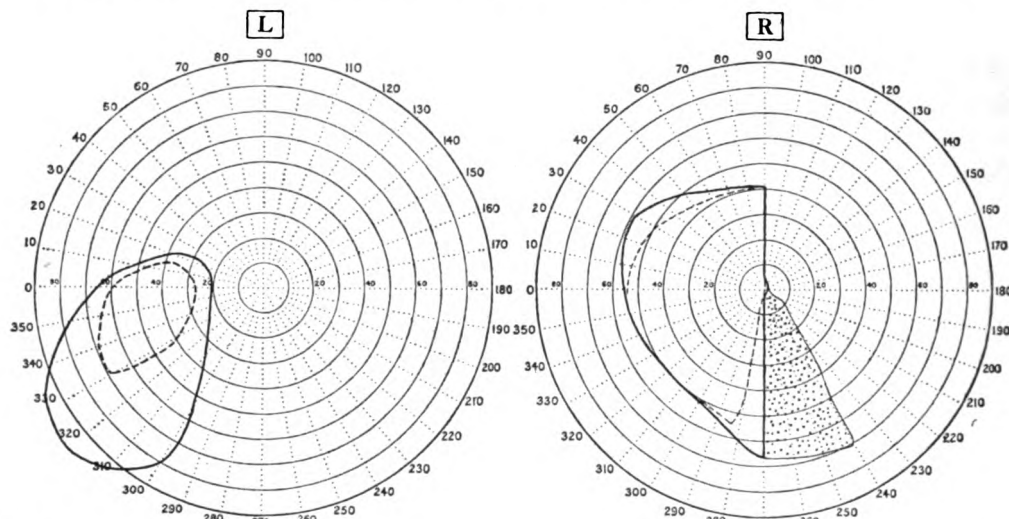


FIG. 185.—BITEMPORAL HEMIANOPIA TERMINATING HOMONYMOUSLY. SAME CASE AS FIGS. 183, 184 FOUR YEARS LATER.

Objects, $2\frac{1}{5}0$, $2\frac{5}{5}0$. Some dim vision still in lower right temporal quadrant for $2\frac{5}{5}0$ (dotted area). (Wnn., 20/11/19.)



FIG. 186.—TUMOUR WHICH CAUSED FIELD CHANGES SHOWN IN FIG. 185. $\frac{2}{3}$ th natural size.

slight or atypical, it is not possible to be sure that true bitemporal hemianopia is present unless the internal isopters show the characteristic temporal deflection, while at the same time they occupy their normal positions in the nasal field. The neglect of this criterion led to the misinterpretation by some observers of the field changes found sometimes in bilateral retrobulbar neuritis and in conditions such as nasal sinus disease and pregnancy. In more developed cases the field changes already described are quite

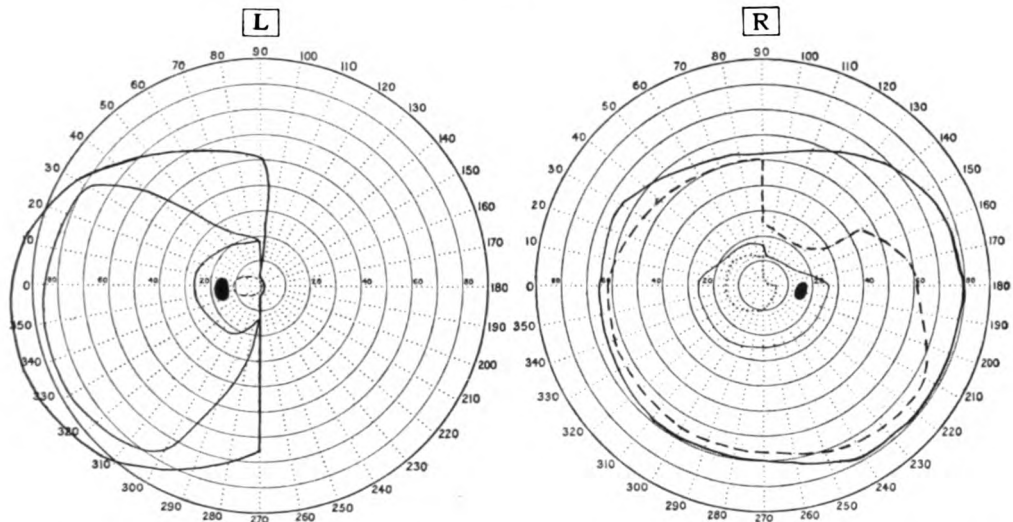


FIG. 187.—HOMONYMOUS HEMIANOPIA FROM LESION AT LEFT ANTERIOR ANGLE OF CHIASMA ON LATERAL SIDE. (Originally regarded as a tract lesion.) Hypophysial tumour with symptoms for about two years. Objects R. $\frac{3}{30}$ —, $\frac{3}{30}$ - - - , $\frac{2}{60}$ —; $\frac{1}{60}$ red (dotted line). L. $\frac{6}{30}$ —, $\frac{3}{30}$ —, $\frac{2}{60}$ —, $\frac{1}{60}$ —... R.V., L.V., $\frac{6}{6}$ +. Pronounced incongruity. The right field is almost normal but for some depression in the upper lateral quadrant. The retained temporal field of the left eye is depressed as is shown by the contraction of the isopters. (Be., April, 1924.)

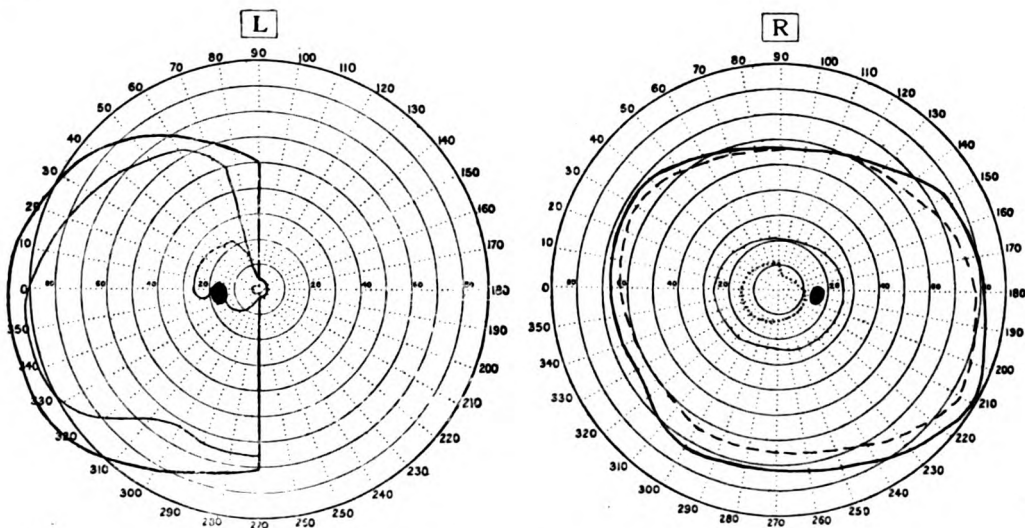


FIG. 188.—SAME CASE AS FIG. 187 FOUR MONTHS LATER (AUGUST, 1924).

The merest trace of hemianopia remains in the right field, shown by slight depression for red $\frac{1}{60}$ in the upper lateral quadrant.

The left field shows increased central depression, but minute central field for $\frac{1}{60}$ remains.

Objects: R. $\frac{3}{30}$, $\frac{3}{30}$, $\frac{2}{60}$; $\frac{1}{60}$ red (dotted line). L. $\frac{6}{30}$, $\frac{3}{30}$, $\frac{2}{60}$, $\frac{1}{60}$. (Be., 1924.)

definite, and no mistake can be made. In order to establish the real nature of these field changes a wide range of visual angles must be used. The apparently normal parts of the field should be tested with angles ranging down to $\frac{1}{2000}$ (1.7'), and the apparently blind parts with angles up to $\frac{40}{330}$, or larger if required. Colours may also

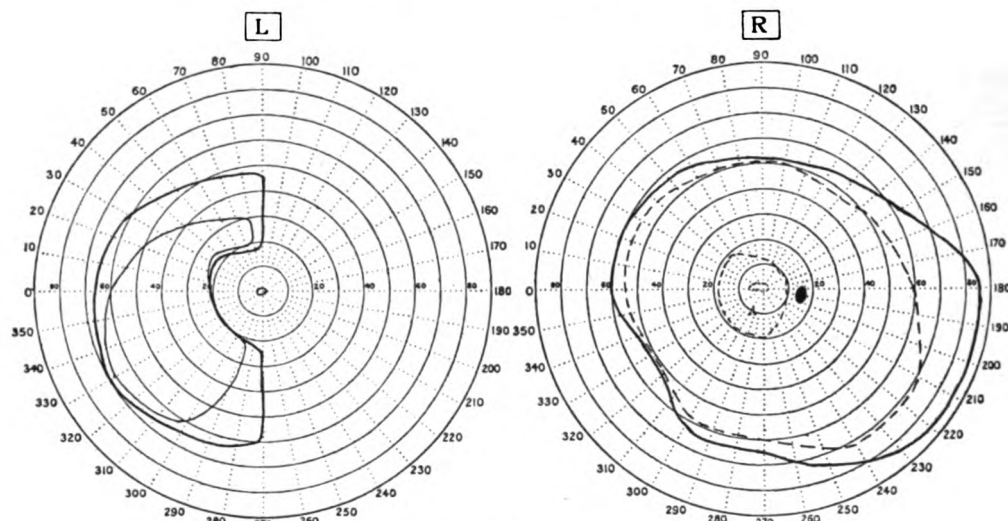


FIG. 189.—SAME CASE AS FIG. 187 THREE YEARS LATER.

The left field is still more depressed, a small central island remains.

Objects: $\frac{1}{3} \frac{0}{3} \frac{0}{3}$, $\frac{5}{3} \frac{5}{3} \frac{0}{3}$, V. $\frac{0}{6} \frac{0}{6}$.

The right field is normal except for a trace of hemianopia in the central area with recognition of red in upper nasal quadrant.

Objects: $\frac{5}{3} \frac{0}{3}$, $\frac{1}{3} \frac{5}{3} \frac{0}{3}$, $\frac{2}{2} \frac{0}{0} \frac{0}{0}$; $\frac{2}{2} \frac{0}{0} \frac{0}{0}$, red (dotted line). V. $\frac{0}{6}$. (Be., 1927.)

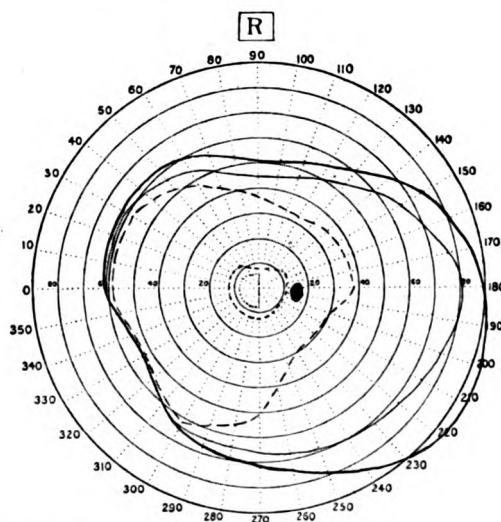


FIG. 190.—RIGHT FIELD OF SAME CASE AS FIG. 189 A YEAR LATER.

The left eye is now blind. The right field shows a trace of hemianopia in the central area and some contraction of the temporal isopters shown by the large size of the objects required.

Objects, $\frac{1}{3} \frac{0}{3}$ —, $\frac{2}{2} \frac{0}{0}$ —, $\frac{1}{3} \frac{5}{3} \frac{0}{3}$ — — —, $\frac{2}{2} \frac{0}{0} \frac{0}{0}$ - - -; $\frac{1}{3} \frac{0}{3}$ red (dotted line).

The left optic nerve is entirely blocked and the crossed fibres of the right optic nerve are all affected. (Be., 1928.)

Two years later the right field, both central and peripheral, had improved.

be used in a similar way, or the room may be darkened if thought necessary. This kind of examination always shows that the chiasmal fibres are much less severely blocked or much more widely affected than is indicated by a single test with a medium size of object, and it also removes any difficulty which may occasionally arise in the recogni-

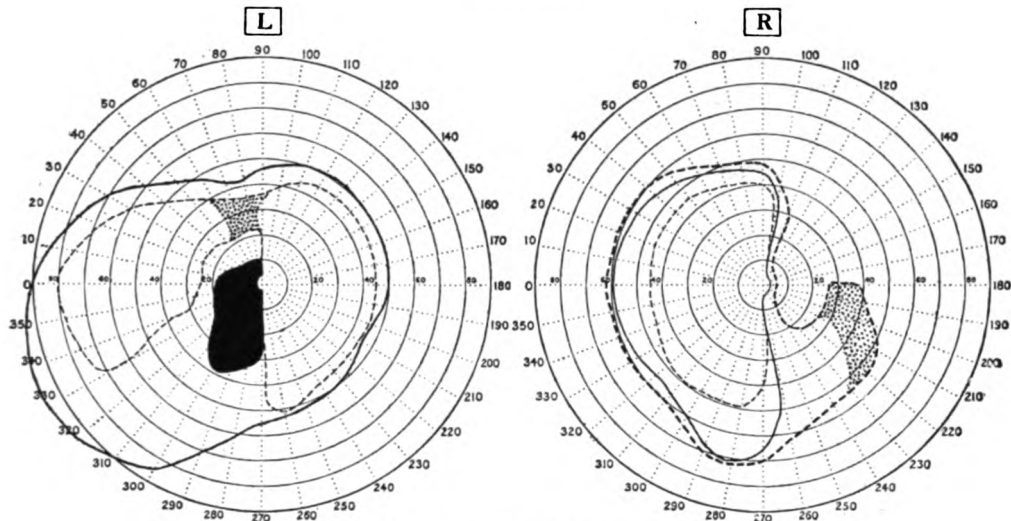


FIG. 191.—BITEMPORAL HEMIANOPIA.

Right field regular. Objects $\frac{3.0}{3.3.0}$, $\frac{5}{3.3.0}$, $\frac{3.3.0}{3.3.0}$. V. $\frac{6}{9}$.
 Left field irregular: Large scotoma breaking through downwards and temporally. Objects $\frac{1.5}{3.3.0}$, $\frac{1}{3.3.0}$. V. $\frac{6}{9}$.
 The dotted parts indicate areas where the test object was dimly seen.
 Symptoms one year. Acromegaly not present. (A., 1922.)

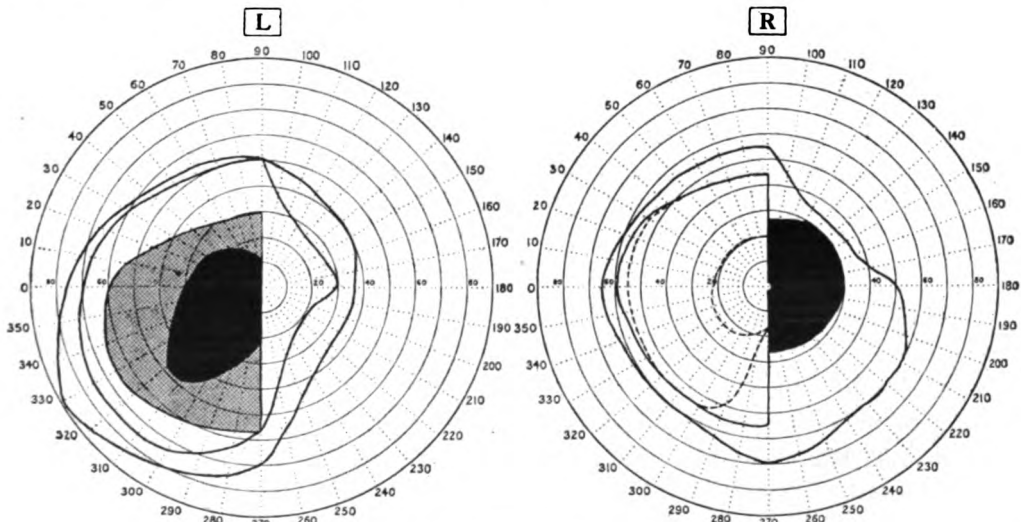


FIG. 192.—BITEMPORAL HEMIANOPIA. TYPE WITH LARGE SCOTOMA.

R. V. = $\frac{6}{24}$. L.V. = hand movements in nasal field. Objects left, $\frac{0.0}{3.3.0}$, $\frac{1.0}{3.3.0}$; right, $\frac{0.0}{3.3.0}$, $\frac{5}{3.3.0}$, $\frac{3.3.0}{3.3.0}$, $\frac{2.0}{3.3.0}$. Eight months' duration, acromegaly not present. Radiograph of sella normal. (B., 1924.)

tion of the bitemporal character of the field changes and enables misinterpretation to be avoided.

In the earliest stages the field changes may be so slight as to offer difficulty in satisfactory demonstration. The statements of the patient with regard to the presence of a blur or mist, especially in connection with reading along a line of print, indicate an exhaustive search for an explanation. No peripheral changes may be demonstrable.

The variation in perception in the different quadrants is very characteristic, though not always present in a high degree and occasionally absent or, at least, undemonstrable. It is usually easily shown on the screen if two or three small white tests—sometimes only one—are used, or if not clearly elicited with white, a coloured object, preferably red, may be chosen. The patient will say that a red object appears red on one side of the field and yellow on the other, or that it appears as white, yellow, pale red, and bright red in the quadrants in the sequence already described if the case is typical and regular. In the earliest stages of the scotoma the object, although recognised as red in the apex of each quadrant, appears of somewhat different shade or tint in the affected apex. These features may be referred to as the “quadrant sign” of a chiasmal lesion. The test should be made quickly, and the patient's first impressions ascertained, for if this form of examination is prolonged the colours become known and in weakly-marked cases no difference may be perceived between the quadrants, or fatigue may cause the colour field to shrink to minute dimensions.

The presence of typical *bitemporal* defects, whether central or peripheral, indicates a chiasmal lesion. The absence of such defects in cases in which a lesion, such as a tumour, in the neighbourhood of the chiasma is suspected on other grounds is no contra-indication; it merely shows that there is no interference with the chiasma. The discovery of a unilateral temporal defect should always be followed by a minute examination of the opposite field. If the defect remains unilateral or develops into a homonymous hemianopia, it is evident that, although the lesion may be near the chiasma, this structure is not affected. Since the changes in one field are always more advanced than in the other, it may be presumed that in the earliest stage one field only is affected, but our knowledge of the earliest field changes in chiasmal lesions is still too incomplete to allow such a conclusion to be regarded as established. The early affection of one field alone is often due to the incidence of the interference being at or near the junction of the chiasma with the nerve or tract rather than in the body of the chiasma.

A prechiasmal tumour situated between the optic nerves may give rise to a unilateral temporal hemianopia becoming bilateral later on, or to blindness of one field with temporal hemianopia in the other, and it is possible that the absence of the temporal island may be indicative of a lesion in this situation. As already pointed out, the existence of bitemporal hemianopia due to a post-chiasmal lesion affecting not the chiasmal body but only the medial sides of both tracts requires confirmation.

(b) To what extent do the fields indicate the part of the chiasma affected?

If the view is accepted that the field changes are produced by obstruction of the circulation, it must be remembered that in the case of a tumour, for example, it is not the position of the tumour but the position of the damage to the circulation which is indicated. Factors such as the course and distribution of the vessels and the relationship of surrounding unyielding structures, as well as the position of the tumour, help to determine the actual site of the conduction interference. For this reason too much reliance should not be placed on inferences from the field changes as to the exact site or position of a tumour or other lesion, though on the whole they may be regarded as a trustworthy guide to the point at which the nerve fibres are affected.

The relation of the field changes to the site of interference may be considered in relation to the fibre architecture of the chiasma (Plate V).

(1) When the interference is median the typical features of bitemporal hemianopia as described are produced. There is reason to believe that in cases in which the scotoma is prominent the incidence of the lesion is relatively posterior, as the macular fibres occupy the posterior part of the chiasma.

(2) Owing to the way in which the crossed fibres from both eyes are spread out over the whole body of the chiasma, a laterally placed interference will involve the posterior crossing fibres of the same side and the anterior crossing fibres of the opposite nerve. At the same time the uncrossed fibres on the side of the lesion will be severely affected while those of the opposite side will escape. In this way it is possible that atypical fields of the diagonal crossed quadrant hemianopia variety may be produced. If one entire half of the chiasma is blocked homolateral blindness with contralateral temporal hemianopia results, but homonymous hemianopia is not produced by a lateral interference with the chiasmal body.

When the interference is at one of the angles of the chiasma the incidence may be from the medial or from the lateral side.

(3) A lesion at the anterior chiasmal angle extending laterally from its medial aspect causes temporal hemianopia on the side of the lesion followed by temporal hemianopia on the opposite side when the lesion has become large enough to extend across the median plane so as to involve the crossed fibres of the opposite nerve (Figs. 156, 158). Further progress leads to blindness on the side of the lesion, as the uncrossed fibres become involved, with temporal hemianopia on the opposite side.

(4) If the incidence is from the lateral side of the anterior angle the uncrossed fibres, along with some of the crossed fibres from the opposite eye (anterior loop or knee of crossed fibres), are affected, giving rise to a nasal hemianopia on the side of the lesion, with a slight temporal defect in the opposite field, an unequal homonymous hemianopia (Figs. 187—190). As the angle of the chiasma becomes more deeply affected blindness on the side of the lesion with relative temporal hemianopia on the opposite side results.

(5) A lesion at the posterior chiasmal angle, if on the medial side, gives rise to temporal hemianopia of the opposite eye, followed by nasal hemianopia on the side of the lesion as the uncrossed fibres become implicated. The lesion advancing into the chiasma also causes a temporal defect in the field of the same side.

(6) If the incidence is on the lateral side of the posterior chiasmal angle, nasal hemianopia on the same side occurs, followed by temporal hemianopia on both sides as the crossed fibres become affected, that is to say, blindness of the first affected eye with temporal hemianopia in the other.

If we summarise these conclusions from the opposite point of view we find that :—

(1) Typical bitemporal hemianopia indicates a median chiasmal interference. Prominence of the scotoma suggests incidence at the back of the chiasma (Plate V. 1).

More advanced loss of the nasal field on one side indicates a lateral deviation of the interference towards that side.

The diagonal crossed quadrant type of field may be attributed to a lateral interference in the chiasmal body.

(2) Temporal hemianopia in one field followed by temporal hemianopia in the other and subsequently by blindness of the eye which was the first to show the field defect indicates involvement of the anterior angle of the chiasma corresponding to the blind eye from the medial aspect (Plate V. 3).

Nasal hemianopia in one field followed by slight temporal hemianopia in the other and subsequently by blindness of the eye which originally showed the nasal hemianopia indicates the incidence of interference at the lateral aspect of the anterior chiasmal angle on the side of the blind eye (Plate V. 4).

(3) Temporal hemianopia in one field followed by nasal hemianopia in the other and subsequently by temporal hemianopia in the field which developed the nasal defect indicates an affection of the posterior chiasmal angle from the medial aspect advancing into the body of the chiasma. The lesion is on the side opposite to the original temporal hemianopia (Plate V. 5). Nasal hemianopia in one field, followed by temporal hemianopia in the other, and subsequently by relative temporal hemianopia in the field originally affected, indicates an incidence on the lateral aspect of the posterior angle of the chiasma extending into the body of the chiasma. The lesion is on the same side as the original nasal hemianopia (Plate V. 6).

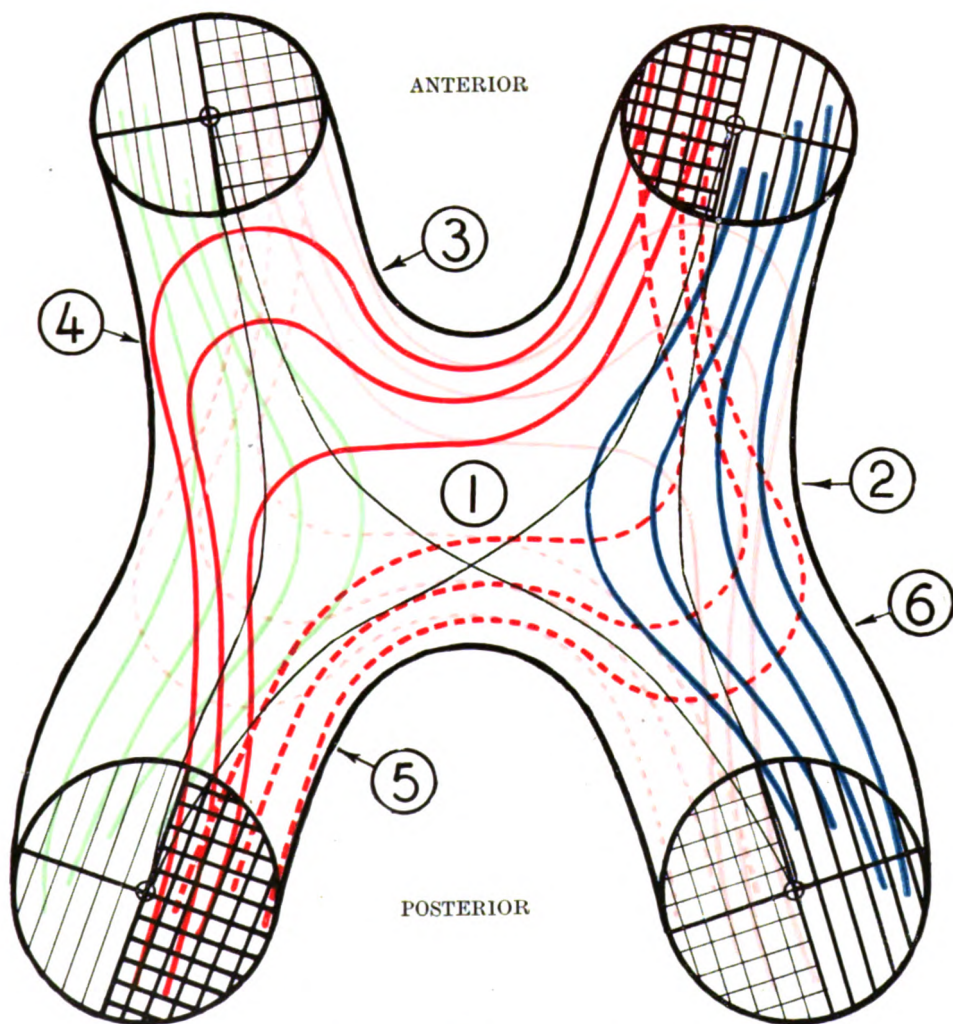
It will be seen that there are four different ways, Plate V., 3, 4, 5, 6, in which blindness of one eye with temporal hemianopia in the other may be produced. They are distinguishable from each other since the sequence of the affected half fields is different. The first two are more common. Except in (5) the eye first attacked is the one which becomes blind.

Binasal hemianopia would require the presence of two lateral lesions acting upon the uncrossed fibres alone, and, therefore, in front of or behind the chiasma proper (see p. 196). According to Wilbrand and Saenger (469, VI. 15) it can only be produced by lesions affecting the lateral sides of the anterior limbs of the chiasma.

When the lower quadrants fail early, producing a more or less regular horizontal hemianopia, pressure on the upper part of the visual path is suggested. This is, no doubt, due to the chiasma being forced upwards against resisting structures, such as the vessels of the *circulus arteriosus*, although more evidence on this point is required. Suprasellar tumours may arise in the region of the hypophysial stalk or of the *tuberculum sellæ* and press upwards the posterior or anterior edge of the chiasma accordingly, producing field changes approximating to the regular type, while less frequently they extend over the upper surface of the chiasma, and cause inferior defects. Persistent defects of the inferior temporal quadrants strongly suggest the presence of a suprasellar tumour.

As already pointed out, too much reliance should not be placed on inferences from the field changes alone as to the exact position of the tumour. Apart from such rare cases as that quoted by Wilbrand and Saenger, in which an occipital and an optic nerve lesion were present at the same time, and produced field defects resembling those due to chiasmal interference, the conditions found at operation or post mortem often vary from what might have been expected from the evidence of the fields. The field

PLATE V.



DIAGRAMMATIC SCHEME OF THE ARRANGEMENT OF THE NERVE FIBRES IN THE CHIASMA. THE NUMBERS CORRESPOND TO THE NUMBERS IN THE TEXT.

[To face p. 234.]

changes may indicate that the chief incidence of the interference is at a certain point, but it by no means follows that the main mass of the tumour or inflammatory product is in that situation. A small knob-like excrescence may project against the chiasma or one of its limbs, though the tumour itself may be either comparatively small or extending in a different direction. This limitation does not mean that perimetry is an unreliable factor in this form of diagnosis ; on the contrary, it is of the greatest value in supplementing, and often in correcting, the information obtained by other methods in conjunction with which it should always be employed.

II. The Cause of the Interference

In the preceding paragraphs the field changes which point to a lesion involving the chiasma and to the part of the chiasma affected have been considered.

Although field defects of bitemporal hemianopic type are most characteristically produced by tumour pressure, other causes give rise to very similar changes.

It remains to discuss how far these field changes help us to ascertain whether the cause is a tumour or some other condition and, if a tumour, what information we can glean with regard to it.

(a) Tumour.

Hypophysial or stalk growths are much the most common, but any form of basal tumour, including aneurysm, may be present. The field changes in all are similar in character. Those which follow the typical and regular sequence are, on the whole, suggestive of intrasellar growths, the more atypical and aberrant changes being more commonly associated with suprasellar tumours or basal swellings unconnected with the hypophysis.

The nature of the cause is indicated by the mode of onset and the behaviour of the field changes which exhibit the characteristic features of pressure defects in other parts of the visual path.

The onset is usually gradual, but rapid or even sudden commencement is recorded. When the history is of rapid onset careful investigation will often show that it is the interference with central vision which has been rapid ; peripheral vision may have failed for some time without attracting the patient's attention. The fields show definite peripheral loss as well as scotomata, and the peripheral loss is a relatively prominent feature. If there is a large and intense scotoma with little and slight peripheral loss the possibility of an inflammatory cause should be considered.

The defects are progressive, though fluctuation and variation are common features but not as rapid or as pronounced as in inflammatory conditions such as chiasmal retrobulbar neuritis.

The persistence and relentless progress of the field changes form the chief indications in favour of the presence of tumour as against other conditions.

The size of the tumour is to some extent reflected in the extent and severity of the field defects, which tend to be slight when the tumour is small and extensive and intense when the tumour is large.

In the case of intrasellar tumours, *e.g.*, hypophysial adenomata, the relation of the

size of the tumour to the first appearance of field defects depends on the height of the chiasma above the diaphragma sellæ. In normal fixation of the chiasma the adenoma must rise 2 cm. (Jefferson 208) before causing visual symptoms by upward pressure against the chiasma. If the chiasma is pre- or post-fixed the enlargement may require to be still greater before field changes are produced.

Severe visual symptoms often indicate intracranial extension of the growth, and, therefore, a large tumour, whereas slight field changes do not necessarily indicate a small tumour, but may be the result of the slow growth of a relatively large swelling. It might be thought, for example, that blindness of one eye with commencing temporal hemianopia in the opposite field would be caused by quite a small lesion at one anterior

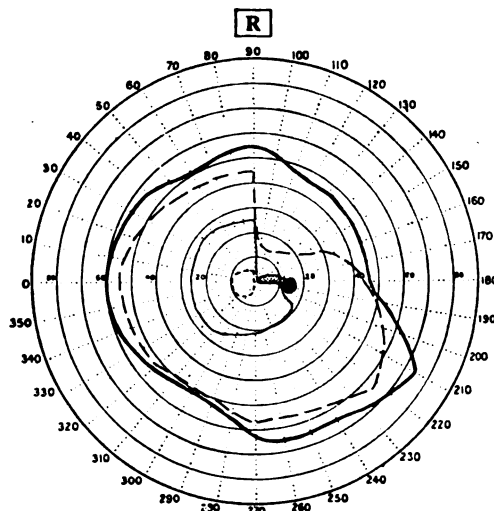


FIG. 193.—BITEMPORAL HEMIANOPIA DUE TO ANEURYSM.

Left eye blind. Right field shows typical changes V. §. (B., 1932.)

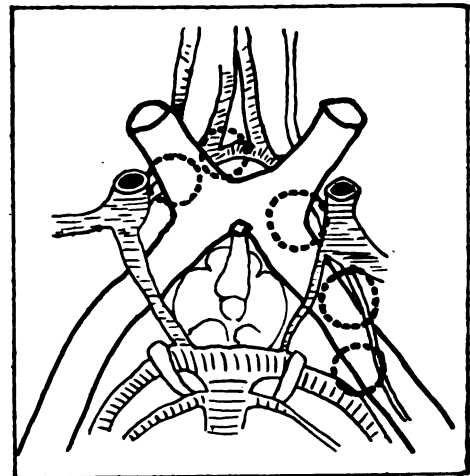


FIG. 194.—COMMON SITES OF BASAL ANEURYSM IN RELATION TO THE OPTIC NERVES, CHIASMA, AND OPTIC TRACTS. (AFTER JEFFERSON.)

angle of the chiasma, but reported cases have been due to large tumours with much distortion of the parts (Walker, Gordon Holmes).

In the early stages the field changes indicate that the tumour has extended sufficiently to touch the visual path, and may be the only or almost the only evidence; later on the presence of the tumour is usually clear apart from perimetry, which provides supplementary information as to its growth and extent.

The scotomatous type of field change is characteristic of activity of the lesion, and the non-scotomatous type of a slowly-growing or stationary condition. The extent and intensity of the scotoma vary with the aggressiveness of the tumour and may diminish during the periods of less active growth, or when the incidence of the interference becomes altered as the structures surrounding the tumour give way here and there and allow the nerve path to be temporarily spared. As there is no sharp division between slowly and rapidly growing tumours, the scotomatous and non-scotomatous types of field change are not separated by any hard and fast line. If a tumour grows sufficiently

slowly, it may attain relatively large dimensions and cause considerable distortion of the optic nerve, chiasma, and tracts, and yet produce only slight visual disturbance.

Difficulty in distinguishing the chiasmal scotoma from others will rarely be encountered, especially if the whole field is examined. Its quadrantic composition is usually easily shown by analysis and a scotoma which does not show this feature should not be regarded as due to a chiasmal lesion unless there is strong additional evidence. This precaution will enable confusion with tobacco amblyopia, Leber's disease, and other forms of bilateral retrobulbar neuritis to be avoided. An isolated central or paracentral scotoma of quadrantic type with normal periphery is exceptional, if it ever occurs, in tumour cases, though it may be present in other conditions.

As in other conditions the steep or gentle slope of the edge of the defect indicates

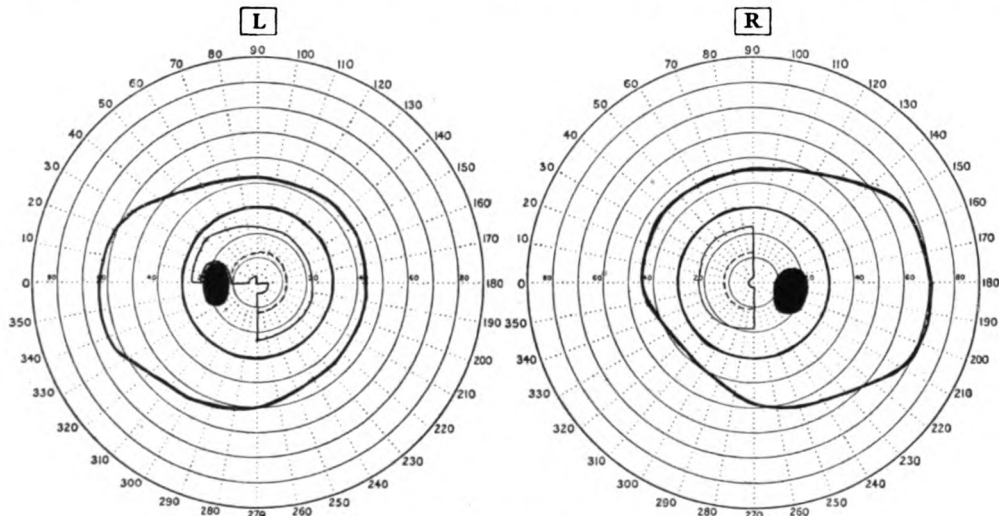


FIG. 195.—BITEMPORAL HEMIANOPIA FROM DISTENSION OF THIRD VENTRICLE.

Cerebellar tumour. Changes mainly central in both fields: lower temporal quadrant defect in left. R.V. $\frac{36}{36}$. L.V. $\frac{18}{18}$. (L., 1935.) (Charts kindly supplied by Mr. Norman Dott.)

the relatively stationary or active nature of the field changes at that part, and the presence of proportion or disproportion provides similar indications. Signs of rapidity of growth suggest the more active types of tumour, but may also be produced by cysts. The rapid or sudden appearance of temporal hemianopia may be due to the formation of an aneurysm near the chiasma or to a hæmorrhage into a hypophysial tumour. Improvement in the field changes indicates that the nerve fibres have not been invaded by the growth, but pressed upon from outside. Sudden improvement or alteration suggests a change in position so as to produce a lessened or new incidence of the pressure. Hard tumours push aside the structures, soft growths tend to envelope the chiasma and its limbs, and are, therefore, more likely to produce atypical field changes.

In regard to the pathological differentiation of the various forms of tumour no direct help is obtained from perimetry alone, but, nevertheless, valuable assistance is gained from the study of the visual fields in association with the whole clinical picture. The field changes give information as to the position and behaviour of the tumours, which,

taken in connection with the patient's age, the X-ray examination, the state of the optic discs, and other circumstances, enable groupings to be made which lead to correct diagnosis in a high percentage of cases. In this way the two main groups of intrasellar and supra- or extra- sellar tumours are easily distinguished. As regards diagnosis, intrasellar tumours give little difficulty, since, as a rule, recognisable X-ray changes are produced before alterations in the visual field. The second group is of special interest to the perimetrist, because of the greater variety of tumours concerned and because in some cases the field changes form the only discoverable deviation from normal health. As the radiographic evidence may be negative the question of differentiation from conditions other than tumour also arises. In this group the most important tumours are meningiomata growing from the region of the tuberculum sellæ and causing slowly

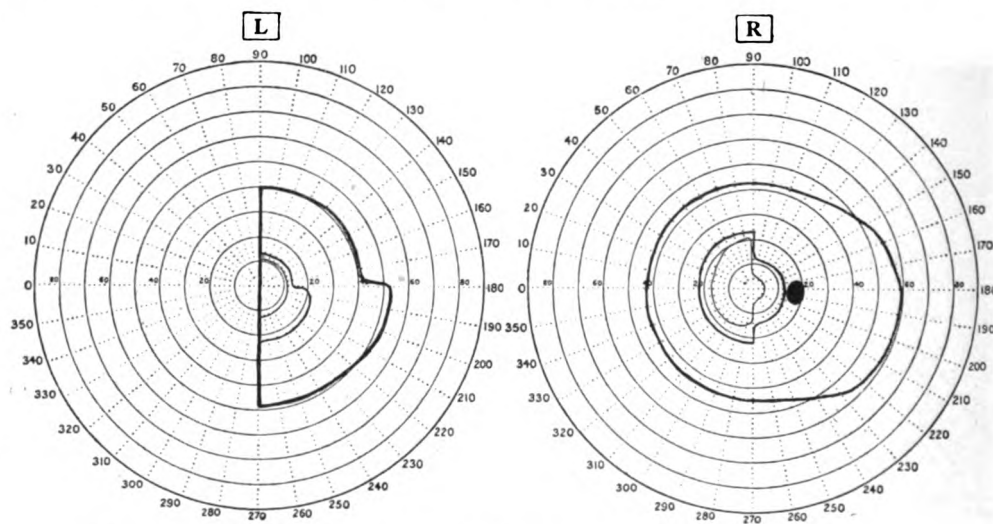


FIG. 196.—BITEMPORAL HEMIANOPIA FROM DISTENSION OF THE THIRD VENTRICLE.

Girl, age 19, with a slow-growing cerebellar tumour. Central parts of field affected in right eye; both central and peripheral in left. R.V. $\frac{6}{24}$. L.V. $\frac{6}{18}$. (J., 1936.) (Charts kindly supplied by Mr. Norman Dott.)

progressing field changes, extrasellar hypophysial adenomata producing more rapidly developing field alterations, cranio-pharyngiomata or Rathke's pouch cysts, and, more rarely, aneurysms and tumours of the chiasma. Pronounced variability in the visual symptoms has been found by some observers to be a feature of aneurysm. Jefferson (207) found the most characteristic field defects associated with aneurysm in the chiasmal neighbourhood to be:— (1) A nasal defect with a scotoma in the field of the same side as the lesion and in the opposite field a homonymous temporal defect. (2) Bitemporal defects commencing with inferior quadrantic loss, usually developing suddenly. (3) A nasal hemianopic defect in the field of the affected side. Chiasmal gliomata are rare in adults and tend to produce somewhat irregular and indefinite hemianopias with relatively great depression of central vision on both sides. Pronounced asymmetry of the fields, particularly such conditions as blindness in one eye and temporal hemianopia in the field of the other, indicating an anterior chiasmal angle interference, is suggestive of meningioma and against hypophysial growth, especially if the sella is normal (Cushing).

In addition to pathological swellings, the physiological enlargement of the hypophysis in the late stages of pregnancy has been stated by some observers to produce bitemporal hemianopia by upward pressure against the chiasma. The fields of vision brought forward in support of this contention do not show the characteristic features of commencing bitemporal hemianopia as caused, for example, by a hypophysial adenoma but merely those of general depression, which, owing to the slope of the temporal field, produces a greater contraction on the temporal than on the nasal side. This form of

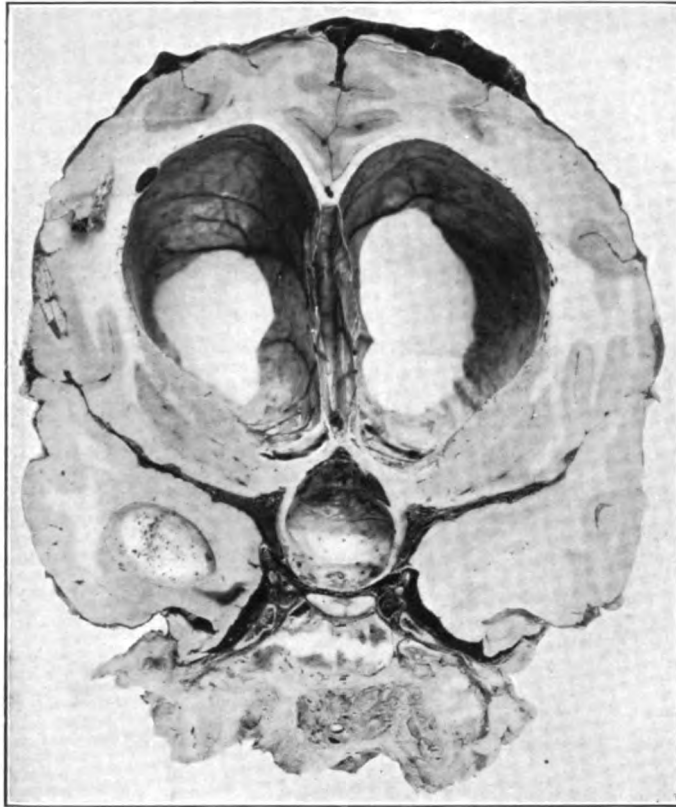


FIG. 197.—HYDROCEPHALUS DUE TO CEREBELLAR TUMOUR, SHOWING HOW THE DISTENDED THIRD VENTRICLE COMPRESSES AND FLATTENS THE SELLA AND HYPOPHYSIS.

Postero-anterior view of coronal section immediately behind chiasma.

(By kind permission of Messrs. Sinclair and Dott and the Editor of the *Transactions of the Ophthalmological Society of the United Kingdom.*) (Sinclair and Dott, 379.)

field change suggests a functional origin. It must be remembered that an intrasellar growth always causes appreciable enlargement of the sella before it produces the earliest signs of bitemporal hemianopia. A pregnant patient may, of course, have true bitemporal hemianopia from a pathological condition, but bitemporal hemianopia present during pregnancy is not the same thing as bitemporal hemianopia due to normal pregnancy without abnormal conditions in or near the chiasma.

Allied to tumour cases are those in which for one reason or another, frequently a cerebellar tumour, the third ventricle becomes so distended that it acts practically as a

suprasellar tumour. Bitemporal hemianopia is produced, without special characters indicative of the third ventricle as the cause, but with relative frequency of inferior quadrant field defects indicating the superior incidence of the interference (Figs. 195, 196).

(b) Causes other than Tumour.

Inflammatory conditions and, in exceptional cases, injury produce field changes resembling those caused by tumours, and it is one of the interesting features of bitemporal hemianopia that the typical sequence of the field changes may be produced by these three different causes.

The most important of these conditions is chiasmal retrobulbar neuritis (Figs. 198—206). The pathology of this disease is similar to that of the ordinary form of acute retrobulbar neuritis as it occurs in the optic nerve and most of the cases may be attributed to multiple sclerosis. Chiasmal cases are probably much more common than existing records show, and in these the presence of bitemporal hemianopic features in the fields clearly indicates the site of the lesion. The disease corresponds in age- and sex-incidence, and in onset and course, to acute retrobulbar neuritis. The field changes are characterised by pronounced central defects and relatively little peripheral disturbance. The defects are definitely chiasmal in type, the quadrant sign being usually well developed in the scotoma, and, as has been pointed out by Roenne, exhibit a pronounced tendency to vary or fluctuate and to "wander" or flit from one part of the field to another.

As in acute retrobulbar neuritis the field changes in this primary chiasmal lesion disappear spontaneously more or less completely, and the immediate prognosis as regards sight is excellent. Repeated perimetry shows that the process of recovery may go on for some time after the patient has ceased to notice any defect of central vision.

Another important, though less common, condition is syphilitic basal gummatous meningitis, which may produce bitemporal hemianopic defects of the scotomatous type. Softenings which may be minute and isolated, due to vascular obstruction usually of syphilitic origin, also occur, and have been found by Wilbrand and Saenger to be the cause of isolated central bitemporal hemianopic scotomata with normal peripheral fields.

Cases associated with chronic local arachnoiditis have been described by Craig and Lillie (65), Bollack *et al.* (39), Vail and others.

This condition known as optico-chiasmic arachnoiditis is associated with multiform field changes, often resembling those of bilateral retrobulbar neuritis with which it may easily be confused. The most common field changes in order of frequency (Bollack *et al.*) are central scotoma, concentric contraction, temporal contraction, nasal contraction, horizontal contraction, and lateral hemianopia. According to Vail a central scotoma with a hemianopic defect of the peripheral visual field, especially if bitemporal, is most helpful and suggestive. The central scotoma appears early and is usually bilateral. It is frequently absolute. There is a tendency for the upper fields to suffer more than the lower ones. The defects alter and flit about in the fields and the visual loss may improve. Although great irregularity of the field edge in cases with concentric contraction is said to be typical there is no characteristic form of field defect. The fields differ from those

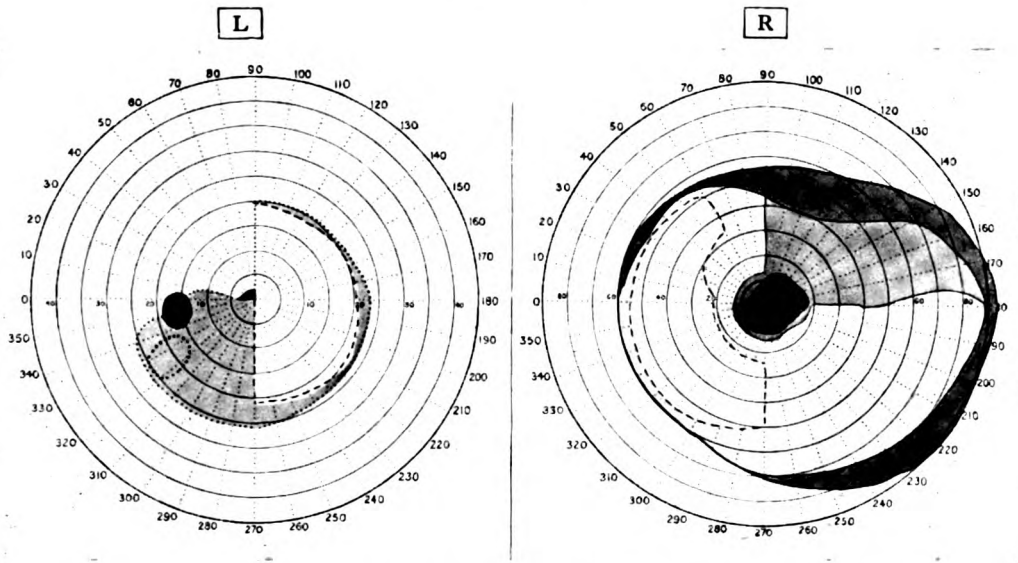


FIG. 198.—BITEMPORAL HEMIANOPIA. CHIASMAL INTERFERENCE WITHOUT TUMOUR. FEMALE, AGE 24. MULTIPLE SCLEROSIS.

On the right side the optic nerve was evidently affected, and the scotoma was too dense to analyse. Objects $\frac{1}{3}$ to $\frac{1}{10}$. The upper temporal quadrant was very dim for $\frac{1}{3}$ to $\frac{1}{10}$. V. = $\frac{1}{10}$ eccentric. L. Periphery normal, central field only charted. Objects $\frac{1}{3}$ to $\frac{1}{10}$ white, $\frac{1}{3}$ to $\frac{1}{10}$ red. V. = $\frac{1}{3}$. The dotted line encloses a strong area for the red test. Note the quadrant scotoma. Duration: right eye unknown, left eye two weeks. (Wy., 16/5/23.)

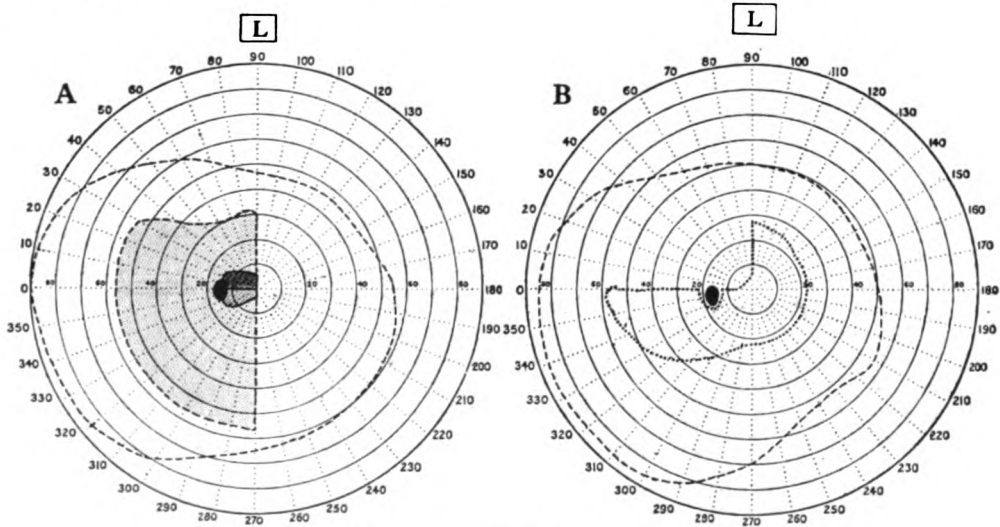


FIG. 199.—LEFT FIELD OF SAME CASE AS FIG. 198.

A. One week later. Left field has developed a large temporal scotoma with more intense central part. Objects, $\frac{1}{3}$ to $\frac{1}{10}$ (periphery), $\frac{1}{3}$ to $\frac{1}{10}$ (scotoma). V. = $\frac{1}{3}$. Right field showed no pronounced change. B. Thirteen days later than A. Periphery normal for $\frac{1}{3}$ to $\frac{1}{10}$, upper temporal quadrant defect for $\frac{1}{3}$ to $\frac{1}{10}$ red. Large temporal hemianopic scotoma now absent. V. = $\frac{1}{3}$.

caused by tumours in so far as temporal hemiachromatopsia has not been described and the approximately symmetrical paracentral temporal scotomata present in tumour cases have not been noted. The loss of vision is more rapid than in tumour cases and the

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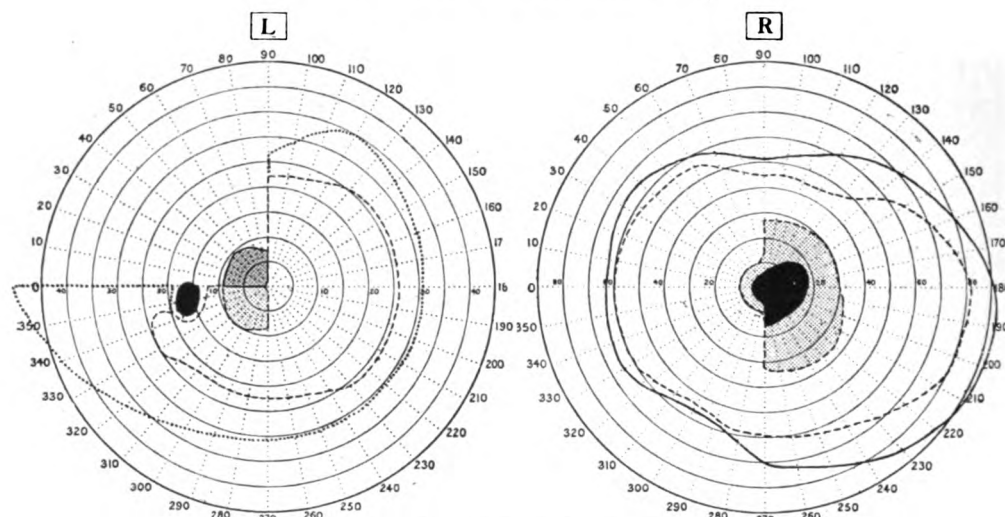


FIG. 200.—SAME CASE AS 198, 199, FIVE WEEKS LATER THAN 196.

Central field of left eye shows upper temporal quadrant colour defect with quadrant scotoma. Objects $\frac{5}{30}$ red; $\frac{2}{000}$, $\frac{5}{000}$ white. V. = $\frac{5}{5}$.
 Right field shows large scotoma partly occupying nasal field. Objects $\frac{5}{30}$, $\frac{1}{30}$. V. = counting fingers at 1 m. (Wy., 20/6/23.)

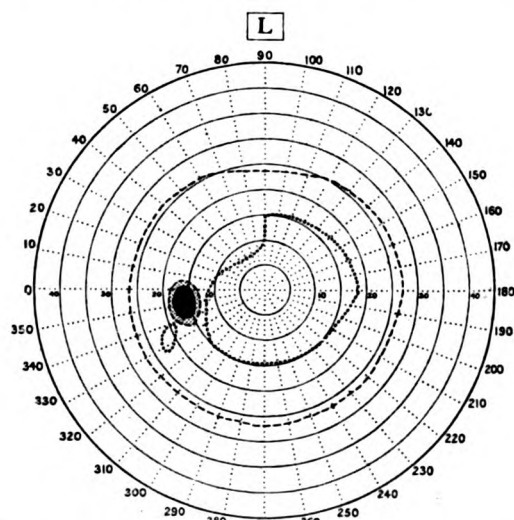


FIG. 201.—SAME CASE AS FIG. 200, NINE MONTHS LATER.

V. = $\frac{6}{6}$. Remains of defect in left field shown by $\frac{1}{300}$ red (dotted line) with a small island for that test. Otherwise field was normal: the $\frac{1}{300}$ isopter only is shown. At this time, apart from a slight enlargement of the circumcaecal amblyopic zone, the right field, originally the more severely affected (Fig. 198), had become quite normal. V. = $\frac{6}{6}$. (Wy., 20/3/24.) Multiple sclerosis developed in 1932.

fields are more irregular and show defects rarely found in tumour cases. The field changes are commonly associated with optic atrophy, less often with papilloedema.

Other local inflammatory conditions, such as tubercle, are exceedingly rare.

Bitemporal hemianopia has been attributed to nasal sinus disease, but a close scrutiny of the published cases weakens rather than supports this view.

In cases of fracture of the base of the skull, bitemporal hemianopia in one of its

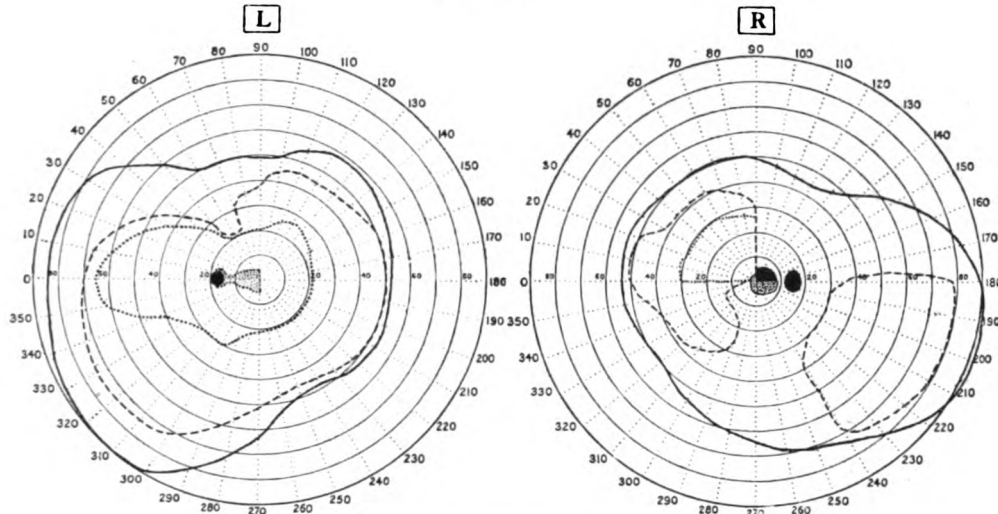


FIG. 202.—BITEMPORAL HEMIANOPIA. FEMALE, AGE 30.

Objects $\frac{3}{3} \frac{0}{0}$, $\frac{1}{3} \frac{0}{0}$; $\frac{1}{3} \frac{0}{0}$ red (dotted line). Red was also seen in the temporal island in the right field, and a relative temporal hemianopia was present for red in the left field. R.V. = Fingers at 3 m. L.V. = $\frac{0}{0}$. (S., 1912.)

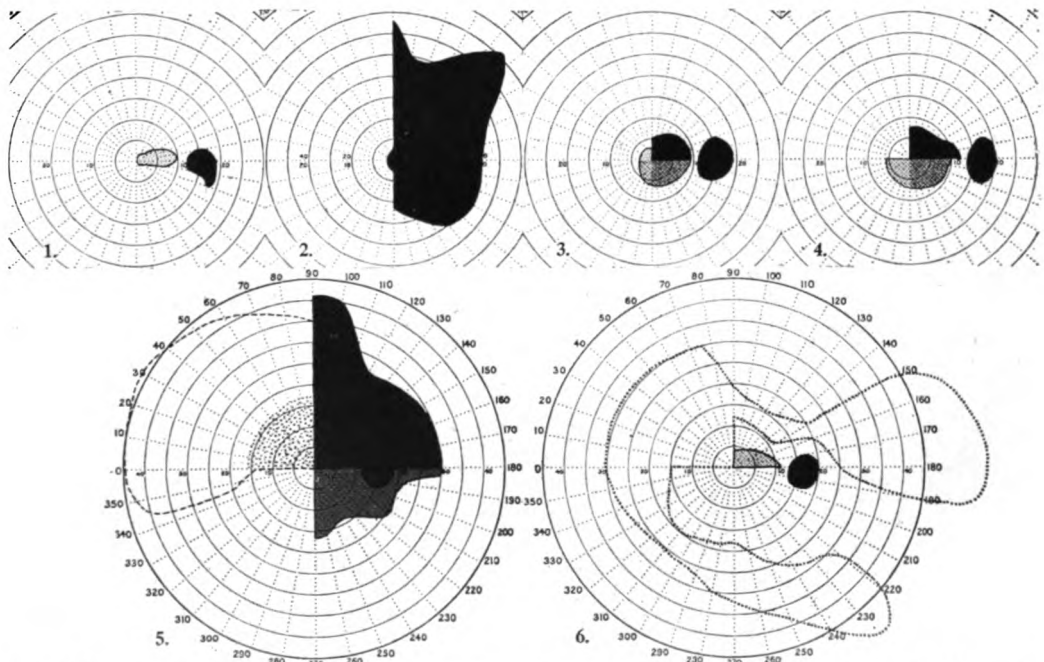


FIG. 203.—THE SCOTOMA IN THE RIGHT FIELD IN THE SAME CASE AS FIG. 202, SHOWING RAPID CHANGES.

(1) August 7th, 1912, $\frac{2}{2} \frac{0}{0}$, $\frac{2}{2} \frac{0}{0}$. V. = $\frac{0}{2}$. (2) August 9th, $\frac{3}{3} \frac{0}{0}$. V. = Fingers at 3 m. (3) August 10th, $\frac{3}{3} \frac{0}{0}$, $\frac{1}{3} \frac{0}{0}$. V. = Fingers at 3 m. (4) August 11th, $\frac{3}{3} \frac{0}{0}$. V. = Fingers at 4 m. (5) August 12th, $\frac{1}{3} \frac{0}{0}$, $\frac{3}{3} \frac{0}{0}$, $\frac{1}{3} \frac{0}{0}$. V. = Fingers at 4 m. (6) August 24th, field for $\frac{1}{3} \frac{0}{0}$ red (dotted line). Scotoma $\frac{1}{3} \frac{0}{0}$ white. V. = $\frac{0}{0}$ eccentric.

stages may be found as soon as the patient can be examined or may arise subsequently, and may be due to direct or indirect damage to the chiasma. Complete bitemporal hemianopia with steep edges, normal colour fields on the nasal side, and sparing of the

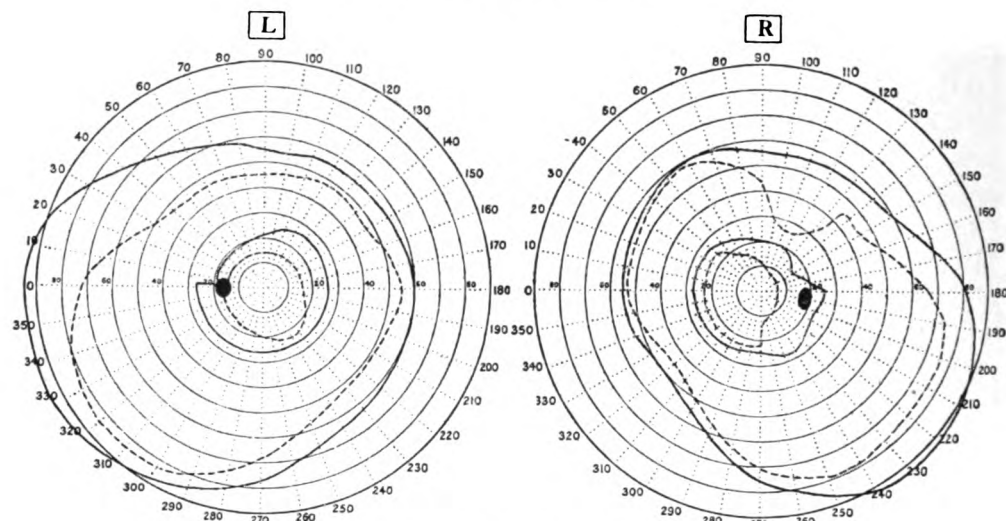


FIG. 204.—SAME FIELDS AS FIG. 202, NINE YEARS LATER.

Traces of hemianopia still remain and in right field a minute temporo-central scotoma (not shown in figure). Objects $\frac{2}{3}00$, $\frac{3}{3}00$; $\frac{2}{0}00$, $\frac{2}{0}00$. R.V., L.V., $\frac{6}{12}$. (S., 1921.)

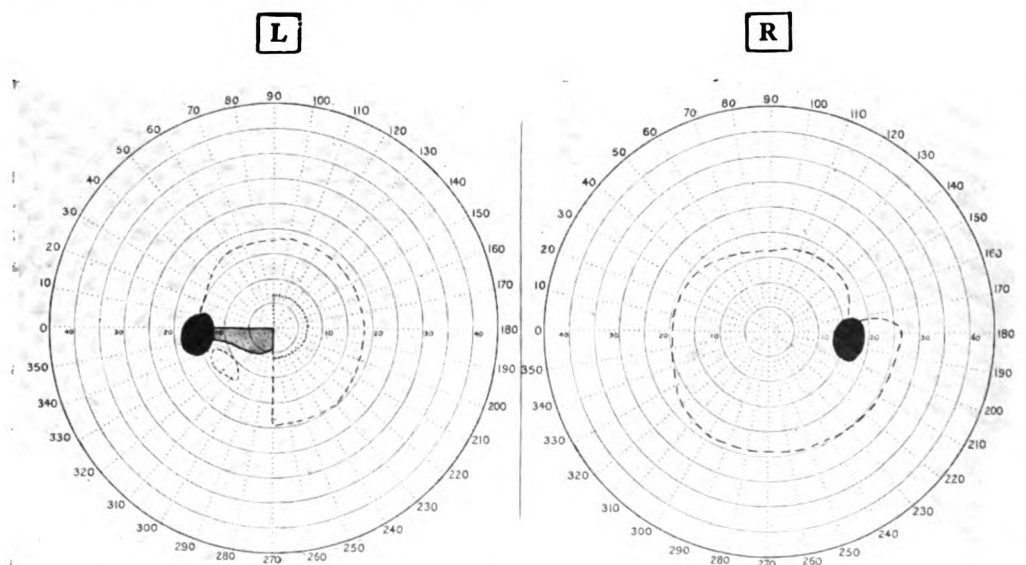


FIG. 205.—CENTRAL TEMPORAL HEMIANOPIA IN LEFT FIELD, MOST PRONOUNCED IN LOWER TEMPORAL QUADRANT, WITH SUSPICIOUS CONTRACTION OF THE $\frac{2}{0}00$ ISOPTER IN THE RIGHT. FEMALE, AGE 21.

Objects: L. $\frac{2}{0}00$, $\frac{2}{0}00$, $\frac{2}{0}00$; $\frac{1}{0}00$ red. R. $\frac{2}{0}00$, $\frac{2}{0}00$. L.V. = $\frac{6}{12}$. R.V. = $\frac{6}{8}$. Recovery in one month, but with traces remaining. (G. 1924.)

fixation area has been recorded. In other cases the field conditions may be exactly similar to those produced by tumour (Hughes, 192), and in one reported by Liebrecht (246) typical field changes, including the temporal island, were present. In another of Liebrecht's cases partial retention of both lower nasal quadrants alone was found, a very rare condition in bitemporal hemianopia, similar to the fields recorded by Hirsch in a case

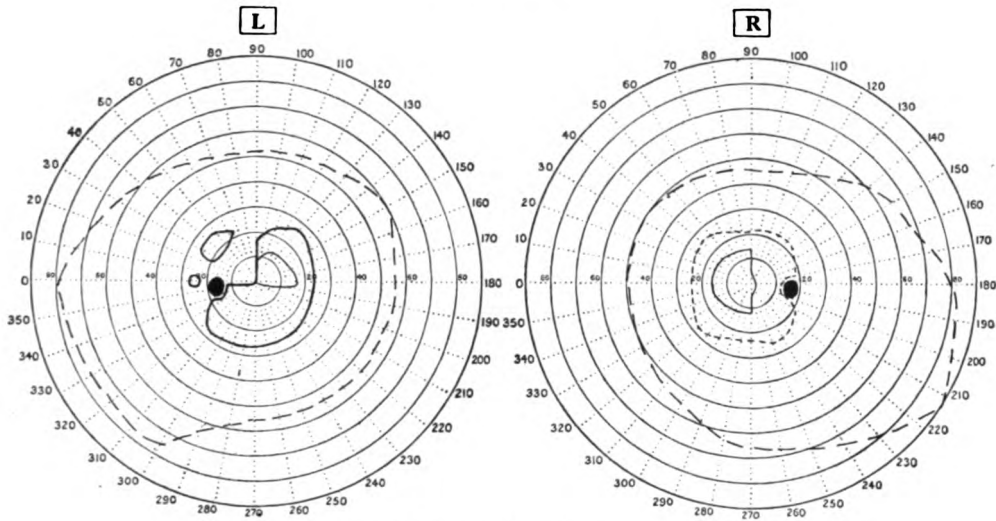


FIG 206.—TRANSIENT BITEMPORAL HEMIANOPIA IN A BOY OF TEN YEARS

Peripheral fields normal. Both central fields show hemianopic changes detected only by small test-objects.
 R.E., V. $\frac{6}{3}$. Objects $\frac{1}{336}$, $\frac{4}{2406}$, $\frac{1}{2006}$; $\frac{1}{2006}$ green.
 L.E., V. $\frac{6}{3}$. Objects $\frac{1}{336}$, $\frac{4}{2406}$, $\frac{1}{2006}$. (R., 1936.)

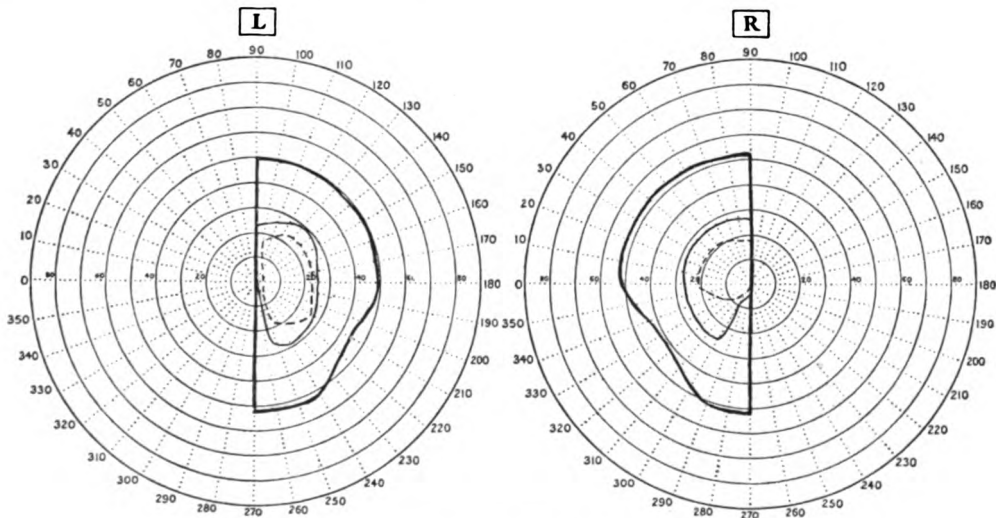


FIG. 207.—BITEMPORAL HEMIANOPIA FOLLOWING INJURY

R.V. $\frac{6}{36}$. L.V. $\frac{6}{36}$. Objects $\frac{1}{336}$, $\frac{4}{2406}$, $\frac{1}{2006}$. (C., 1934.)

of hypophysial tumour. These features show that the crossed fibres were not entirely destroyed, and suggest that the field changes were caused not purely by traumatic rupture of the chiasma itself, but largely by damage to the membranes and blood-vessels followed, in the slowly developing cases, by cicatricial processes which produced interference with the nutrition of the nerve fibres.

Uthoff has reported instances of chiasmal injury with bitemporal hemianopia and others with horizontal hemianopia, the fixation area being divided in each case. In

several cases Cushing (70) made a median antero-posterior section of the chiasma with resulting clear-cut bitemporal hemianopia with divided fixation area.

In diagnosis the chief difficulty is that of distinguishing between inflammatory conditions and the group of extrasellar tumours with negative radiographic evidence.

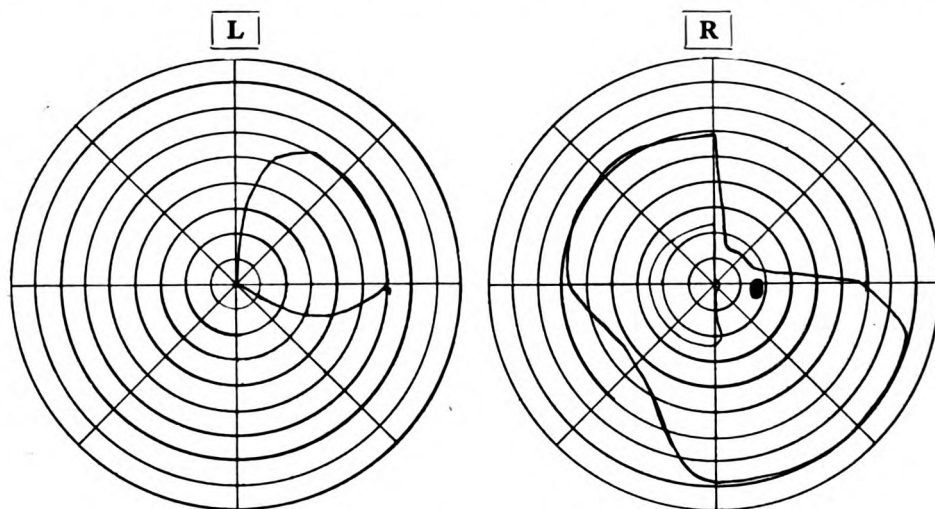


FIG. 208.—BITEMPORAL HEMIANOPIA FOLLOWING INJURY.
Hughes (192). Courtesy of Editor and Publisher of *Trans. Ophth. Soc. U.K.*

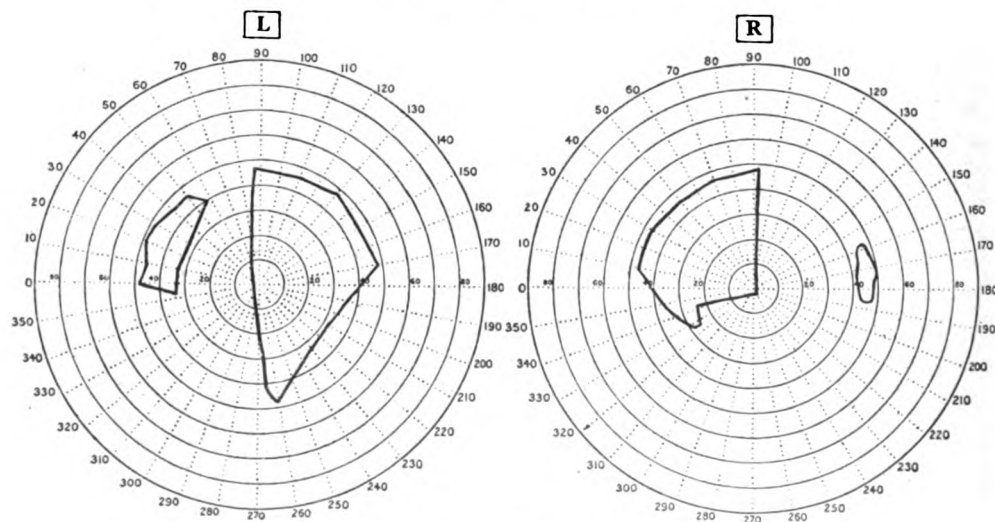


FIG. 209.—BITEMPORAL HEMIANOPIA DUE TO INJURY.
(After Liebrecht.) Note presence of temporal islands and retention of upper nasal quadrants.

The answer to the question: "Is a tumour present or not?" depends more on the behaviour and the relative intensity of the field defects than on the other characters such as shape and position. Rapid and fluctuating changes, with relative prominence of the scotoma and comparatively little involvement of the peripheral field, suggest inflammation, while slower, more progressive and more persistent defects, with pro-

nounced peripheral loss, point towards tumour. Care must be taken to exclude the more persistent forms of bilateral retrobulbar neuritis of the optic nerves by a close search for quadrantic features in the field defects.

Of the inflammations, chiasmal retrobulbar neuritis is the more common and is distinguished by its activity and the age and sex of the patient. Syphilis is less common and more persistent, and may lead to gross field defects and even blindness. It is therefore less easily differentiated from tumour.

In any case of bitemporal hemianopia perimetry is obviously only one of the many sources of evidence upon which the diagnosis is based, and although the field changes may be so characteristic as to be almost pathognomonic, caution should be exercised before basing conclusions as to the nature of the lesion upon perimetry alone. In most cases other evidence can be elicited, but in a few the visual changes form the only ascertainable deviation from normal. Symptoms of hypophyseal disturbance in association with bitemporal field changes do not necessarily point to hypophyseal tumour, nor does a normal sella turcica without hypophyseal disturbance exclude tumour in that neighbourhood.

Prognosis.—The prognosis depends upon the nature of the cause as well as upon the state of the field changes and their behaviour and course. The same indications which assist in forming the diagnosis, afford some help in regard to prognosis and treatment. The natural tendency of the vision and of the field defects to fluctuate or alter in progress or behaviour should be remembered. The significance of the nature of the edge of the defect and of the presence of disproportion has been mentioned. Stationary or improving fields should not be regarded as indicating a similar condition of the tumour or attributed to the effects of treatment, other than surgical, without caution and delay. Improvement in the fields indicates only a diminution in the interference with the visual pathway, not necessarily a reduction in the size of the tumour. Nor is it necessary to infer from a history of progressive visual loss that this will increase; it is from the character of the fields at any stage as well as from the mode of attainment of that stage that deductions as to further visual loss may be drawn. Rapid onset and progress of the field changes suggest a rapidly growing tumour and are often unfavourable signs, but phases of this kind may alternate with periods of comparative or actual quiescence.

While some tumour cases terminate fatally within a few years or even less, in others the growth appears to become stationary, so that the patient may live and work with comparative comfort and facility for many years even though with only one useful eye. If there is still persistence of the temporal island or perception of large objects in the defective parts of the fields, improvement may be expected after a successful operation.

As regards the position of the tumour and the possibility of its removal, we have seen that some indications are afforded by the field changes. Blindness of one eye with temporal hemianopia in the other field usually indicates an anterior and more accessible position of the tumour. The prognosis for the blind eye is poor, but the eye with temporal hemianopia may recover completely. Homonymous hemianopia, on the other hand, is, on the whole, an unfavourable sign as it often depends on a posterior position or extension of the growth.

A change from bitemporal to homonymous hemianopia indicates that an alteration in the relationship of the tumour to the chiasma has occurred, and may initiate a period of improved vision. It does not necessarily indicate cessation of growth of the tumour. The state of the fields has, therefore, a certain influence on the question of the advisability of operation and on the precise procedure (frontal or trans-sphenoidal approach) best suited to each individual case. For these reasons a careful perimetric study should precede the consideration of operation, and the character of the fields should be included in the evidence on which the decision is based, and should be interpreted with care and deliberation, without, of course, claiming for it any predominant importance.

After a successful operation the fields retrace the stages through which they originally developed. In this way the significance of the early stages is confirmed.

In the majority of syphilitic cases improvement in the field under appropriate treatment is to be expected and indicates a favourable prognosis, but secondary processes of a cicatricial nature may cause serious and permanent damage, so that hopes based on early improvement may be disappointed.

CHAPTER XI

SUPRACHIASMAL PATH AND VISUAL CORTEX

THE symptomatology of local disease implicating the visual path between the eye and the tracts, though not purely ophthalmic, is comparatively free from evidence of involvement of neighbouring nerve structures. Behind the chiasma the visual fibres become closely related to other nerve tracts and centres, and the diagnosis of the situation of lesions above this level becomes correspondingly dependent upon signs of interference with adjacent parts of the brain.

Still more posteriorly after emergence from the internal capsule the visual path becomes less closely associated with nerve structures, interference with which gives rise to obvious symptoms, until in the occipital region lesions again often produce field defects unaccompanied by other symptoms.

The extent of the suprachiasmal path and visual cortex, and their liability to damage, suggest that close examination of the symptoms produced should be of value for exact diagnosis, and in perimetry the means are at hand for measuring and recording functional impairment with considerable delicacy and accuracy. Although only one kind of field change, homonymous hemianopia, occurs, perimetry not only can give much information as to the nature of the involvement of the visual path, but also may suggest lines along which further examination should be pursued. The importance of perimetry in this respect has been conclusively demonstrated by Uhthoff, Cushing and Walker, and others, and its advantages and possibilities are becoming more fully appreciated.

The development of the field changes may commence at an early stage before the appearance of diagnostic or localising indications of other kinds, and may not attract the notice of the patient, while in other cases, visual symptoms are the first to which his attention is drawn. The field changes may be the only evidence of disease present, or they may develop during the course of the illness when their appearance marks a stage in its progress. In order to obtain the full advantages of perimetry, it is, therefore, necessary to make the examination periodically and to watch for early signs.

In testing for hemianopia it is often useful to begin by examining the binocular field with the confrontation method. The patient is asked to maintain fixation with both eyes on the face of the examiner who stands 4 or 5 ft. in front of him. The examiner then holds up both hands simultaneously and asks the patient whether he sees two or one, whether the fingers are open or closed, moving or stationary. Or the screen may be rapidly used, testing both eyes at once. In both cases it is often desirable to conduct the examination in reduced illumination. In doubtful or difficult cases, especially in the earlier stages of tumour, there may be only an occasional and variable hemianopic depression so elusive as to render accurate charting impossible. It may be impossible to map out the blind spot or to obtain satisfactory results by the perimeter or screen, or a degree of mental dulness, depending on the general condition of the patient, may be

present. Under such conditions an intelligent application of the confrontation method, together with close attention to the patient's remarks and behaviour, will often prove more successful than other forms of examination. A modification of this method is employed by Norman Dott as an "attention test." The examiner stands in front of the patient and places one hand at the extreme temporal periphery of each field. The hands are gradually moved forwards and towards the median plane, the forefingers being given one single and rapid twitch occasionally, until first one and then both movements are seen. The interval, if any, is noted. The patient's attention becomes attracted to the stronger image and the other, though visible, escapes notice at first. Dott has found this method more useful than tests of each field separately in cases of low-grade hemianopic defect, especially if the patient is ill and examination at the screen therefore difficult.

If nothing can be discovered by the confrontation method each field should be tested very thoroughly using every means to elicit very faint defects by the methods already described. The central area should be tested on the screen rather than with the perimeter.

A negative finding with a rough test only shows the absence of gross hemianopia and should not be allowed to stand in the way of a more adequate investigation.

In cases of suspected cerebral tumour the fields should be carefully examined by methods for the detection of defects of very slight intensity as the patient may be quite unaware of any visual symptom. Each field should be tested separately by the perimeter in case a defect of the temporal crescent might be present. Occasionally this is the first symptom of homonymous hemianopia. The examination should be continued with the screen using small test-objects both coloured and white.

Once the presence of homonymous hemianopia has been established further examination is directed towards ascertaining its characters and its significance.

In the perimetric study, the onset and behaviour of the hemianopia form important features, and afford guidance not less valuable than that provided by its mere presence.

The assistance afforded by perimetry forms an item in the evidence, upon which diagnosis is based, the value of which, though less in some cases than in others, is always considerable, and, with further development, promises to become still more important. In discussing diagnosis, although the field changes only are referred to, it is not intended in any way to suggest that an unduly prominent position in the clinical picture should be assigned to them.

The nomenclature of hemianopia has already been briefly considered (p. 59); the terms subgeniculate and geniculo-calcarine or suprageniculate, in reference to the anatomical site of the causal lesion, may be added here. That field defects due to lesions of the suprachiasmal path must be essentially homonymous in character, although the changes themselves may differ in the two fields in regard to their onset, position, extent or intensity, is evident on anatomical grounds.

Homonymous hemianopic fields which are superimposable and identical, are congruous, those in which the defects are dissimilar are incongruous. Genuine incongruity on an anatomical basis has a certain diagnostic value, inasmuch as it may be found when the interference with the visual path occurs at a point where the fibres from

corresponding retinal points, or minute areas according to Wilbrand, have not yet come together, that is to say, in the tract, at least in its lower part.

A second variety of incongruity depends upon the normal difference in size between the nasal and temporal fields, the latter being the larger by a peripheral crescentic area corresponding to the unpaired part of the binocular field. A defect of suprachiasmal origin may, therefore, be present in this part of one temporal field without being represented in the opposite nasal field, and constitutes a unilateral partial hemianopia in the form of a temporal crescent or, as it has been termed, the temporal or peripheral half moon (Behr, 26). On the other hand, this crescentic area may be preserved, while the remainder of the temporal field up to the vertical meridian is defective, in association with the complete nasal field of the opposite eye (Fig. 227). It is evident that unilateral hemianopia of supragenulate origin can only be partial. As regards the anatomical basis, it is believed that the most medial fibres of the optic radiation correspond to the nasal periphery of the retina and terminate in the most anterior part of the visual cortex, which is therefore connected with crossed fibres only. It is possible that a lesion may involve—or may spare—this cortical area or its fibres alone, but careful quantitative examination is required to show the true nature of a defect which suggests this origin.

In diagnosis the presence of the temporal crescent is more important than its absence.

The dissimilarity may be due to errors of observation. Even the most experienced observer, dealing with the most intelligent and practised subject, cannot always avoid a certain margin of error which is especially liable to occur in relative hemianopia, unless a series of test-objects is carefully used, since the field boundaries may not be at all well defined. Such errors may be greatly increased if the patient be tired or inattentive, or the observer hurried or careless, or if the technique employed be in any way unsound or inadequate. Special care is required not to allow any shifting of position of the head when the examination is transferred from one eye to the other. The results should be confirmed by testing both eyes together when, if incongruity is present, the outline of the larger field should be obtained. In incomplete hemianopia rotation of the head may produce a slight degree of apparent incongruity.

Other forms of incongruity whose nature is not yet fully established have also been observed (see Roenne, 341; Wilbrand and Saenger, 469, Bd. VII.). Apparently incongruous supragenulate hemianopia calls for the most thorough and exhaustive examination before correct inferences can be drawn.

Sparing of the fixation area, which has already been referred to in connection with chiasmal lesions, also occurs in affections of the tracts and radiations, and is the rule in occipital hemianopias. It is less extensive, less complete, and more frequently absent in subgenulate than in supragenulate, especially occipital, hemianopia. A rare form is the overshoot field, in which the sparing is not only central, but extends along the whole vertical meridian. Explanations such as bifurcation of the macular fibres or bilateral cortical representation of each macular area do not seem acceptable.* The simplest view is that either the sparing is genuine, the nerve elements concerned

* See Appendix III., p. 295.

having partly or wholly escaped injury or possessing more than one source of vascular supply (Lister and Holmes, 257), or it is merely apparent and due to a habit on the part of the patient of fixing slightly to one side of the fovea in order to obtain the best use of his central vision. This is probably not infrequently done by patients who have had hemianopia with a divided fixation area for some little time, and may be held to account for some of the narrow sparings of about half a degree, which are often found in chiasmal and tract hemianopia when carefully tested on the screen. Obliquity of the dividing line corresponds to the relationship of the true vertical meridian of the retina to the distribution of the crossed and uncrossed fasciculi. A spurious form may, of course, be produced if the patient's head is not level during the test, and is associated with vertical displacement of the blind spot. To ensure accuracy with regard to the character of the sparing and the relationship of the edge of the field to the vertical meridian, a careful examination on the screen at 2 m. or more using several test-objects of different sizes, or in reduced illumination, should be made. Care should be taken that the head is exactly vertical. Each field should be examined independently and then both together. Unless there is incongruity the three fields should correspond. Androg  (5) insists that no statement can be made about sparing of the fixation area unless the field has been examined with small visual angles, $\frac{1}{2000}$ or even less, using pilocarpin to contract the pupil so as to eliminate diffusion at the edge of the test-object. He believes that sparing, when it exists, is found equally in subgeniculate and suprageniculate lesions.

In certain cases the defect may cross the vertical meridian and invade the seeing half-field. This occurs in tract hemianopias when the chiasma or opposite tract becomes involved, and occasionally in occipital lesions such as tumour or h morrhage, by pressure on the opposite occipital lobe. Bilateral lesions may also occur, producing bilateral homonymous hemianopia either simultaneous or in sequence, and the defect in one side of the binocular field may differ considerably from that in the other.

Homonymous hemianopia may be relative or absolute, or the intensity may vary in different parts of the defect or at different times. The hemianopia may begin suddenly or gradually, and may remain permanent or may recover partially or completely.

Thus different combinations of several variable factors, such as the extent, intensity or position of the defect or defects in the two fields, lead to the production of many different field pictures all essentially of homonymous hemianopic type, and all dependent on the anatomical position and severity of the causal lesion or lesions. On these lines Wilbrand and Saenger have enumerated no less than thirty varieties of homonymous hemianopia.

Without attempting to follow this plan, and with these few preliminary remarks, we may now proceed to examine the chief features of the field changes produced by lesions in the different parts of the suprachiasmal path.

OPTIC TRACT

The tract is seldom the primary site of disease, probably on account of its small size and sheltered situation, but may become involved in or affected by disease of the adjacent parts. Of the conditions which affect the tracts in this way by far the most

common is tumour; inflammatory conditions such as basal syphilis and multiple sclerosis are much less frequent, while vascular disease and injury are rare. Aneurysm of the circulus arteriosus may occur. The general features of tract hemianopia are those of the bitemporal form, but with homonymous defects as if one field in a bitemporal hemianopia were laterally reversed. Incongruity is the characteristic feature, and is almost always present whatever the cause except in advanced cases. Scotomata and the gourd-shaped or uncinat deformation of the temporal field, so common in bitemporal hemianopia, are rare, especially if the site of interference is some distance behind the chiasma. The field changes begin usually in one quadrant and are relative, incomplete, incongruous and progressive. The edges are sloping and the fixation area is often spared at first. The incongruity may be considerable; examples such as a quadrant defect in one field with either little or no defect or well-developed hemianopia in the other are frequent. As the interference extends and becomes more severe the field defects enlarge and become more intense and less incongruous, while the fixation area becomes affected. Complete interruption of the tract leads to complete and absolute homonymous hemianopia with vertical bisection of the fixation area. This is, however, an uncommon result, since tract hemianopia is usually due to tumour, which tends to involve the posterior angle of the chiasma and therefore to modify the purely homonymous character of the fields. In this way also the macular fibres become affected, since they cross at the posterior part of the chiasma. These features characterise the subgeniculate type of homonymous hemianopia in contrast to the geniculo-calcarine type, in which the macular fibres are nearly always preserved. A complete division of the tract is, of course, indistinguishable on perimetric findings alone from a complete division of the visual path at a higher level.

The course of the defects corresponds to the cause. When this is pressure, as from a tumour or some similar condition, the onset is usually gradual and the defects slowly increase. In inflammatory conditions the onset may be sudden and recovery rapid.

Interpretation.—The patient may complain of visual symptoms and the hemianopia may be pronounced and obvious, or the case may be one in which, in the absence of any complaint on the part of the patient, the possible presence of hemianopia is suspected. In the latter case a negative finding should only be accepted after a very thorough examination, using visual angles as small as $\frac{1}{2000}$ if necessary, as the early indications may be very slight in intensity and confined to a small sector of the field.

The next point to determine is the site of interference, whether sub- or supragen-iculate.

When the onset is gradual, the course progressive, and the edge of the defective area sloping, incongruity between the two fields suggests a tract lesion, as a partial hemianopia due to a supragen-iculate lesion practically always presents defects similar in extent and intensity. The appearance of indications of progressive involvement of one or both of the primarily unaffected half-fields is important as indicating extension to the chiasma or opposite tract, and thus locating the original interference in the anterior part of the tract. In such cases blindness of the eye on the side of the lesion, with temporal hemianopia in the opposite field, may follow an initial homonymous

hemianopia. A lesion at the anterior angle of the chiasma may produce field changes having a similar character but different order of incidence (p. 233). A superior quadrant hemianopia indicates an inferior incidence of the interference and vice versa. Should the hemianopia appear first on the temporal side of the field, or be more developed on that side, the interference may be referred to the medial aspect of the contralateral tract; should it be first or greater on the nasal side the site of interference is at the lateral aspect of the homolateral tract as in the case of temporal lobe tumours. Cushing, on the other hand, regarded the hemianopia of temporal lobe tumours as suprageniculate, *i.e.*, as evidence of direct interference with the optic radiation as it passes through the temporal lobe in Meyer's loop.* A defect commencing in an upper temporal quadrant suggests a lesion lying between the tracts and indicates the necessity for a very careful examination of the opposite field.

The presence of a central hemianopic scotoma in a homonymous hemianopia which is otherwise suggestive of tract lesion, also points towards this site of interference as such scotomata are very uncommon in suprageniculate hemianopias. Division of the fixation area occurs more frequently when the lesion is situated in the anterior than in the posterior part of the suprachiasmal path and strongly suggests the former situation, but this feature alone cannot be relied upon as a localising symptom.

Sudden and complete hemianopia, whether the fixation area is divided or not, cannot be ascribed to a tract lesion on perimetric grounds. Similarly sudden but incomplete hemianopia, especially if stationary, offers difficulty unless there is incongruity. As a rule in such cases the lesion may be presumed to be suprageniculate unless there is evidence to the contrary, and it is necessary to make sure of the nature of the incongruity by careful quantitative examination. Lesions of the posterior part of the tract cannot be located with certainty on perimetric evidence alone if unaccompanied by symptoms indicating the involvement of adjacent structures.

Tumour

Tumours seldom originate in the tract itself, though sarcoma, gumma, tuberculoma and cysticercus are recorded. Interference is usually due to a swelling in the neighbourhood, commonly hypophysial, more rarely connected with some other basal structure. The majority of such tumours lie between the tracts behind the chiasma, though occasionally the lateral side of a tract is compressed by a hypophysial enlargement. Tumours of the temporal lobe affect the tract, first of all on the outer side, by pressure, occasionally by invasion. Subgeniculate homonymous hemianopia may also be produced by tumours of the frontal lobe, especially in its postero-inferior part, of the optic thalamus, and of the corpora quadrigemina.

The interference may, therefore, come from either the medial or the lateral side, and may affect the anterior or the posterior part of the tract. In the former case the chiasma also may become involved, in the latter the adjacent basal ganglia and nerve paths.

Owing to the fact that at this level the fibres from corresponding retinal points have not yet completely come together, a sufficiently small lesion in one tract, especially

* See Appendix III., p. 294

in its anterior part, may affect one field only, and incongruity is common, indeed characteristic. A gradually encroaching lesion produces gradually increasing field defects.

Thus interference with the lower part of the tract produces superior homonymous quadrant hemianopia in which the earlier or worse affected sector corresponds to the tract fasciculus which is sooner or more severely damaged.

Apart from its homonymous character, tract hemianopia due to tumour resembles in its general features the bitemporal form, with the differences already mentioned. The onset is usually gradual. The defect is relative at first, and begins as a partial depression of one quadrant, or sometimes as a lateral failure. Its edge is sloping in the early, but usually steep in the later stages. One field may be affected before the other, and usually exhibits a more advanced degree of development. Progress may be slow or rapid, steady or fluctuating, but uniform and practically absolute visual failure in the affected half-fields, as in occipital hemianopia, is rarely met with in the first instance, since it requires the complete interruption of the tract. Thus considerable disparity as regards the position, extent and intensity of the changes may exist between the two fields. This incongruity in homonymous hemianopic fields is a prominent and important feature and is very suggestive of tract lesion. Finally, the homonymous half-fields may be completely obliterated, but before this stage is reached the field picture may become complicated by involvement of the chiasma or sometimes of the opposite tract.

Scotomata, when they occur, are similar in character and behaviour to those of bitemporal hemianopia. The scotoma may occupy the apex of the most affected quadrant or may appear to be more or less independent of the peripheral field changes, and may be relative or absolute.

The fixation area is spared to a greater or less extent unless the conductivity of the tract is totally destroyed or a central scotoma is present. The sparing may be relative, that is, present for large objects, absent for small objects or colours.

Lillie (254) found that in tumour of the lateral part of the transverse fissure complete clear-cut homonymous hemianopia was present already at an early stage. The hemianopia is complete because the visual path at this level is concentrated in the tract, and its appearance before the development of other symptoms due to involvement of the basal ganglia or internal capsule constitutes a sign of diagnostic value, since in other tumours in this area the hemianopia is a relatively late feature and may be incomplete as in the quadrant hemianopia of temporal lobe tumours.

We have seen that the involvement of the anterior part of the tract by a lesion primarily affecting the chiasma is indicated by the development of the homonymous type of hemianopia in addition to or in place of a previous bitemporal type. In the former case blindness of the eye on the side of the affected tract with temporal hemianopia in the other field results, a significant though not pathognomonic indication, since it may also result from multiple lesions. Accurate diagnosis requires that the development of the process should be watched by periodic examinations. The *replacement* of a bitemporal by a homonymous defect strongly suggests a tumour which is obtaining room by extending backwards into the interpeduncular space, thereby relieving the pressure on the chiasma.

Interference with both tracts is nearly always due to tumour lying between them or to syphilis, and has already been referred to in connection with bitemporal hemianopia. The involvement of the second tract is usually a relatively late develop-

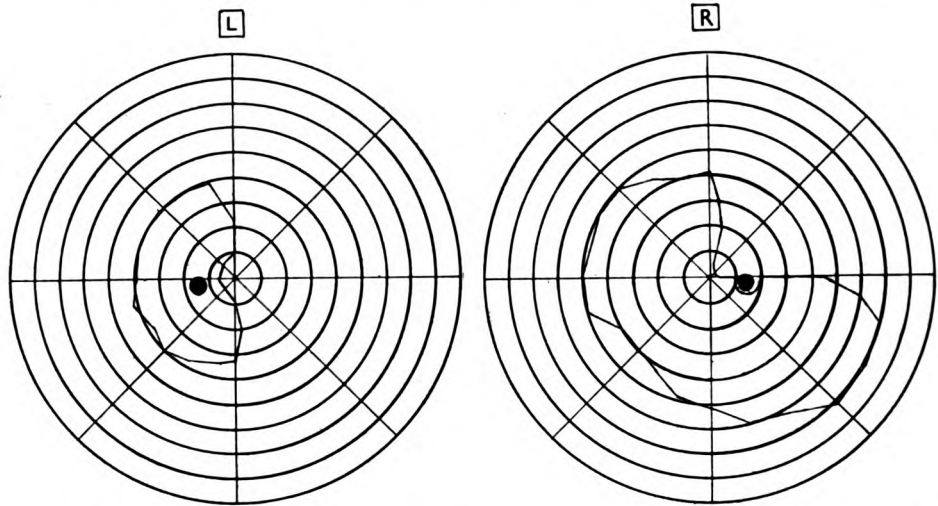


FIG. 210.—HOMONYMOUS HEMIANOPIA FROM HYPOPHYSIAL TUMOUR (Walker and Cushing, 454), SHOWING PRONOUNCED INCONGRUITY.

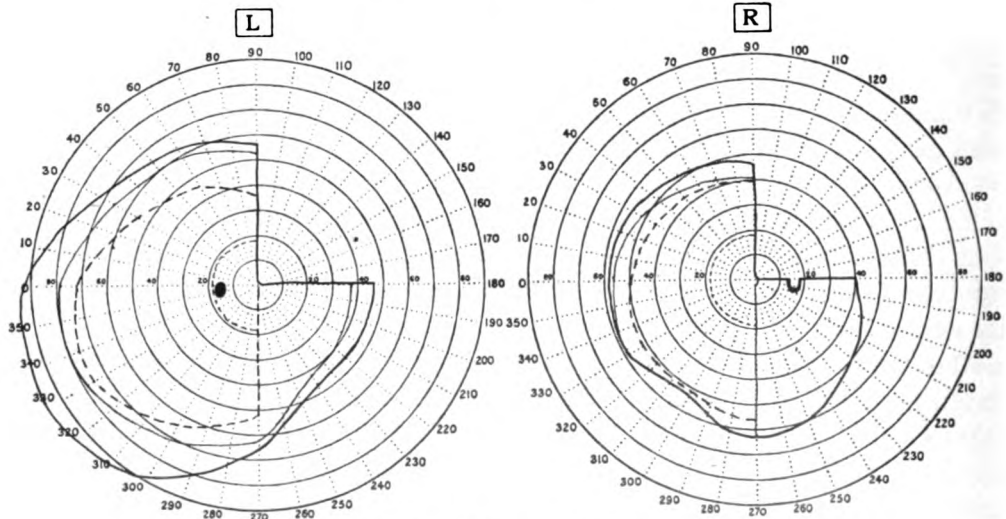


FIG. 211.—HOMONYMOUS HEMIANOPIA FROM TRACT LESION.

Aneurysm at junction of carotid and ophthalmic arteries. In the lower temporal quadrant of the right field $\frac{5}{330}$ was not seen and $\frac{2}{330}$ only dimly. Objects $\frac{2}{330}$, $\frac{5}{330}$, $\frac{8}{330}$. R.V. $\frac{1}{2}$. L.V. $\frac{3}{4}$. (W., 1929.)

ment and produces a defect in the previously unaffected temporal field in an already well-developed homonymous hemianopia, leading to blindness of one eye with temporal hemianopia in the other (see p. 233), and ultimately to complete blindness. Bilateral tract interference without chiasmal involvement is rare, if it ever occurs.

When the interference is on the lateral side of the tract as in temporal lobe tumours,

the defect appears in the nasal field of the eye on the side of the lesion. For detailed charts of the field changes in temporal lobe lesions, we are indebted to Cushing. Although regarded by him as due to radiation, i.e., suprageniculate, interference, the characters of

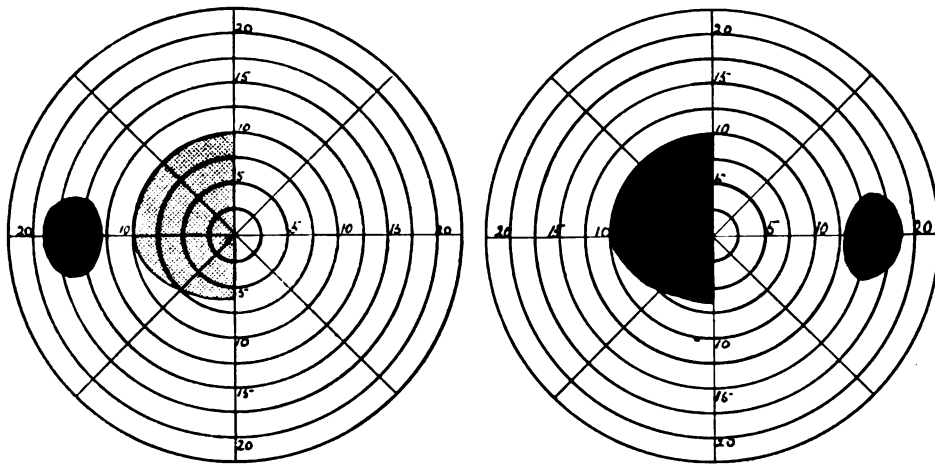


FIG. 212.—LEFT HOMONYMOUS HEMIOPIC CENTRAL SCOTOMA CONGRUOUS IN SHAPE AND EXTENT, INCONGRUOUS IN INTENSITY.

A fortnight later the scotoma became incongruous in extent also. Peripheral fields normal. Scotoma absolute on right side; present for $\frac{2}{3}$ white and $\frac{1}{3}$ red on left side. R.V. $\frac{3}{4}$. L.V. $\frac{1}{4}$. Large tumour of right frontal lobe interfering with lower part of right tract. Recovery after operation with much improvement in scotoma. In the left field the scotoma disappeared. (P., 1942.)

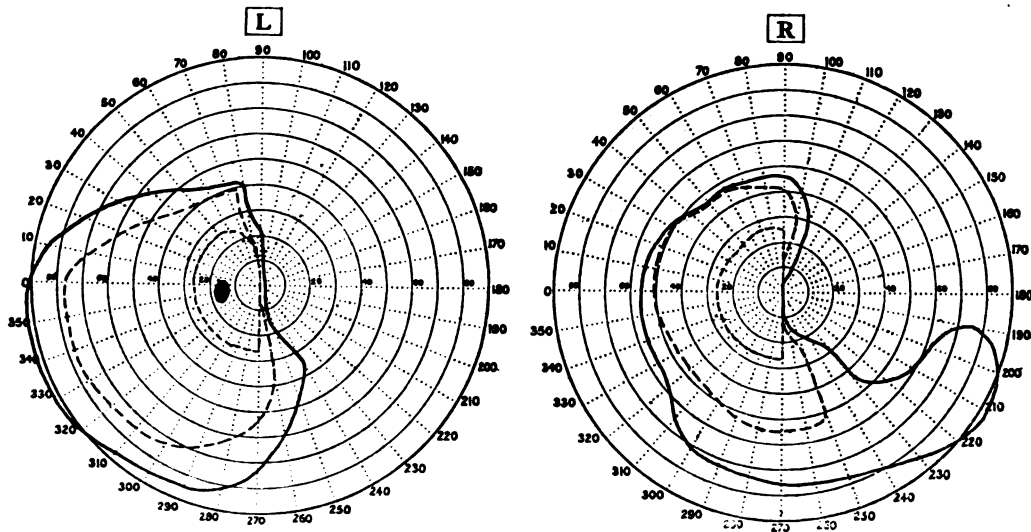


FIG. 213.—HOMONYMOUS HEMIOPIC, INCONGRUOUS, REFERABLE TO TRACT INTERFERENCE: NEUROLOGICAL DIAGNOSIS TUMOUR OF MIDBRAIN

Objects $\frac{1}{2}$ white, $\frac{1}{2}$ red; $\frac{1}{2}$ part; L.V. $\frac{1}{2}$. Fixation area divided for small object. (Pa., 1923.)

the field changes are those of tract, i.e., subgeniculate, hemianopia. Incongruous homonymous quadrant hemianopia without central scotoma, developing relatively late in the sequence of symptoms, is the characteristic feature. As in the case of commencing bitemporal hemianopia from hypophysial tumour, the earliest change may be demon-

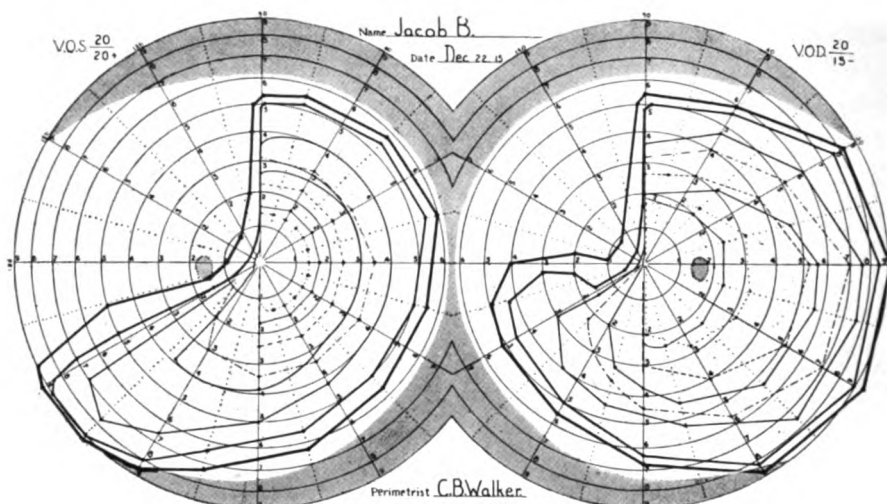
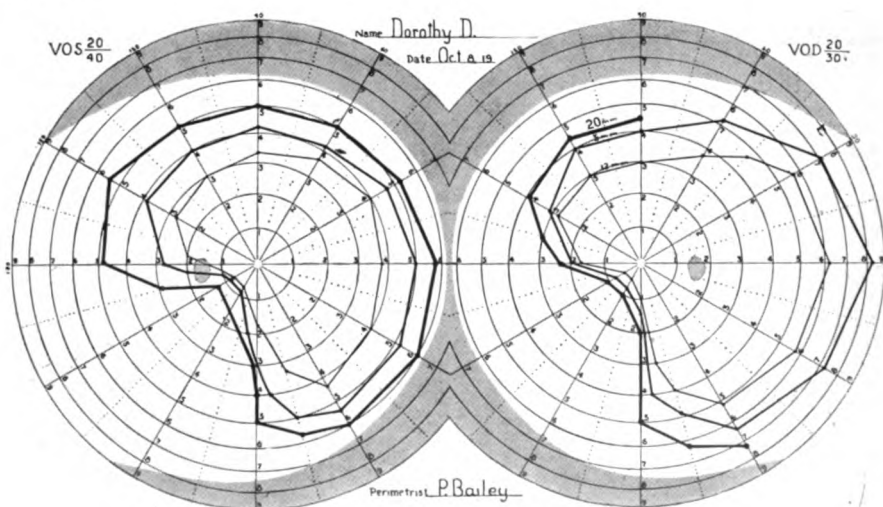


FIG. 214.—FIELD CHANGES IN TUMOUR OF TEMPORAL LOBE.

Cyst in lower part of right temporal lobe. Incongruous hemianopic defects on side of field opposite lesion. Cushing. *Brain*, Vol. 44, 1921.



Before operation

FIG. 215.—FIELD CHANGES IN TUMOUR OF TEMPORAL LOBE.

Gliomatous cyst in upper part of right temporal lobe. Cushing. *Brain*, Vol. 44, 1921 (69).

strable towards the central part of the field, while the periphery is still apparently normal. In its early stages the hemianopia is partial, has sloping margins, and tends to recover if the pressure is relieved. Incongruity is characteristic and may be extreme, one field showing a well-developed quadrant defect while the other is normal. The hemianopia later becomes complete with ultimate division of the fixation area, and, in the final stages, its characteristic features may be swamped by the general visual loss due to secondary optic atrophy. Hemianopic central scotoma is rare, probably owing to the position of the macular fibres in the tract and the arrangement of their blood supply and

to the usual gradual incidence of the pressure as these tumours tend to grow slowly. In the majority of cases the upper nasal quadrant is first affected, but when the lesion is high in the temporal lobe the lower nasal quadrant is the first to become impaired (Figs. 214, 215).

With careful perimetry hemianopic defects should be elicited in over 50 per cent. of temporal lobe tumours at some period during their course.

Diagnosis.—The onset, course and intensity of the defects give some assistance towards the diagnosis of the nature of the condition present in the same way as in affections of the chiasma.

Relative defects commencing slowly with sloping edges in the earlier stages and

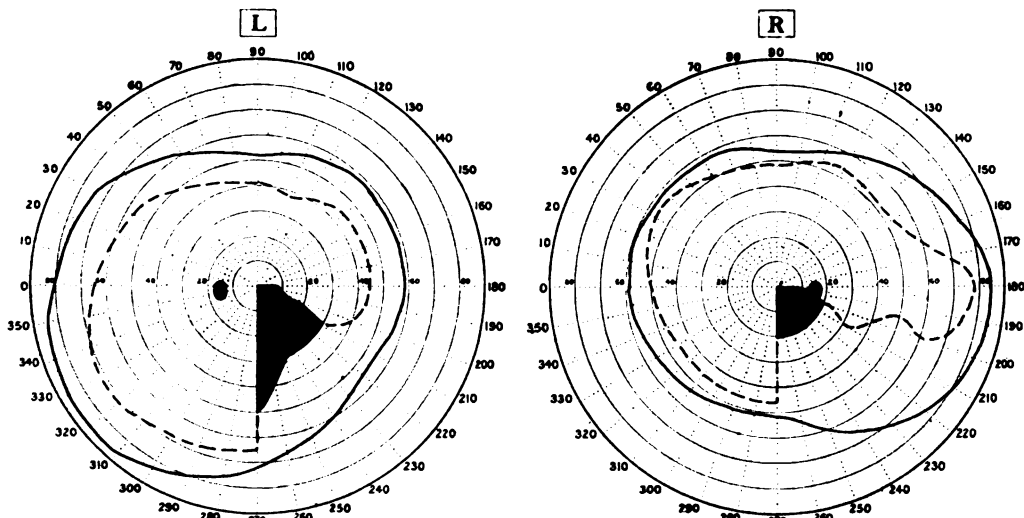


FIG. 216.—HOMONYMOUS QUADRANT HEMIANOPIA WITH SCOTOMA. FEMALE, AGE 36. Fields for $\frac{1}{2}$, scotoma for $\frac{1}{2}$. Periphery normal for $\frac{1}{2}$. Sudden onset, rapid and complete recovery. R.V. $\frac{1}{2}$. L.V. $\frac{1}{2}$. Neuritic focus in tract. (Hg., 1921.)

gradually increasing in extent and intensity until complete hemianopia is produced indicate interference due to pressure.

Basal tumours expanding between the tracts often produce an upper temporal quadrant defect in the contralateral field as the first sign owing to interference with the inferior crossed fibres in the lower portion of the tract. As the interference spreads through the tract and reaches the uncrossed fibres in its infero-lateral part an upper quadrant defect in the field on the side of the lesion is added producing an incongruous hemianopia, with the greater defect in the contralateral field, which subsequently becomes complete. At the same time, or earlier, the chiasma itself becomes involved leading to impairment of the previously unaffected half-fields.

On the other hand the hemianopia of temporal lobe tumours usually appears first in the upper nasal quadrant on the homolateral side since the infero-lateral aspect of the tract is affected. As the whole thickness of the tract becomes involved an incongruous quadrant hemianopia, with the greater defect in the field on the side of the lesion, develops and may progress to complete hemianopia, but the previously unaffected

half-fields do not become impaired unless by depression due to secondary atrophy of the optic nerves.

Johnson (214) found that the homonymous hemianopia caused by frontal lobe tumours was frequently complete whereas in temporal lobe tumours it is usually incomplete.

Aneurysms are indistinguishable from tumours on perimetric evidence alone, except sometimes by the course of the field defects.

The presence or absence of sparing of the fixation area is not in itself a reliable differentiating feature between subgeniculate and geniculo-calcarine hemianopias.

Multiple Sclerosis

The lesions of multiple sclerosis affect the tracts in the same way as the optic nerves or chiasma and produce field changes which resemble in behaviour those of

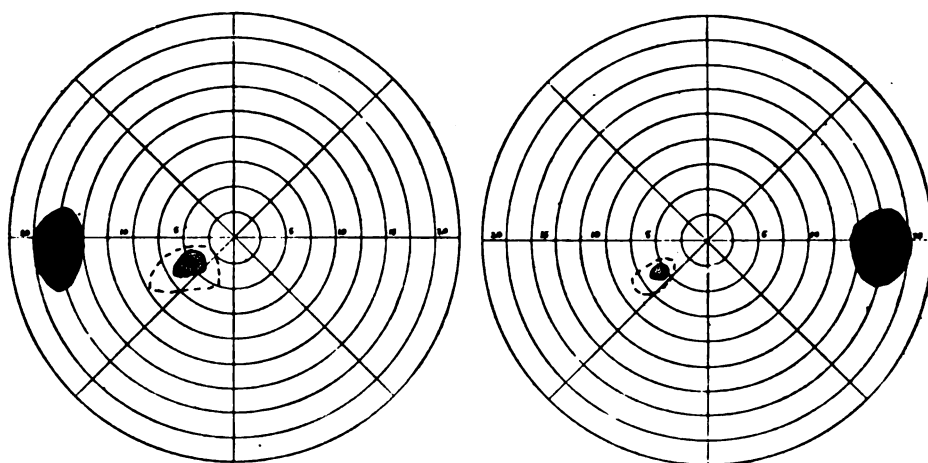


FIG. 217.—HOMONYMOUS INCONGRUOUS PARACENTRAL SCOTOMATA. FEMALE, AGE 24.

Sudden Onset. Associated with Symptoms of Multiple Sclerosis. R.E., scotoma for 20° , nucleus for 20° . V. 2° . L.E., scotoma for 20° , nucleus 20° . V. 2° . (H., 1939.)

ordinary acute retrobulbar neuritis. Homonymous hemianopic defects, varying in extent from small hemianopic central scotomata to the loss of a quadrant or of half the field occur, and central vision is involved. Combinations of different forms may be found. Apart from their hemianopic character these field changes do not differ in onset or course from those described under multiple sclerosis affecting other parts of visual path, and the diagnosis and prognosis are based upon the considerations already discussed. A sudden or rapidly developing homonymous quadrant defect, for example, may be difficult to explain at first, but if it occurs in a young woman and soon disappears, multiple sclerosis is strongly suggested (Figs. 216, 217).

Syphilis

The neighbourhood of the intracranial portions of the optic nerves and chiasma is a favourite site for gummatous meningitis. An isolated tract lesion of this kind is rare, more often the chiasma and one or more of the cranial nerves in the neighbourhood are also involved. The field changes resemble those caused by tumour, but, as in

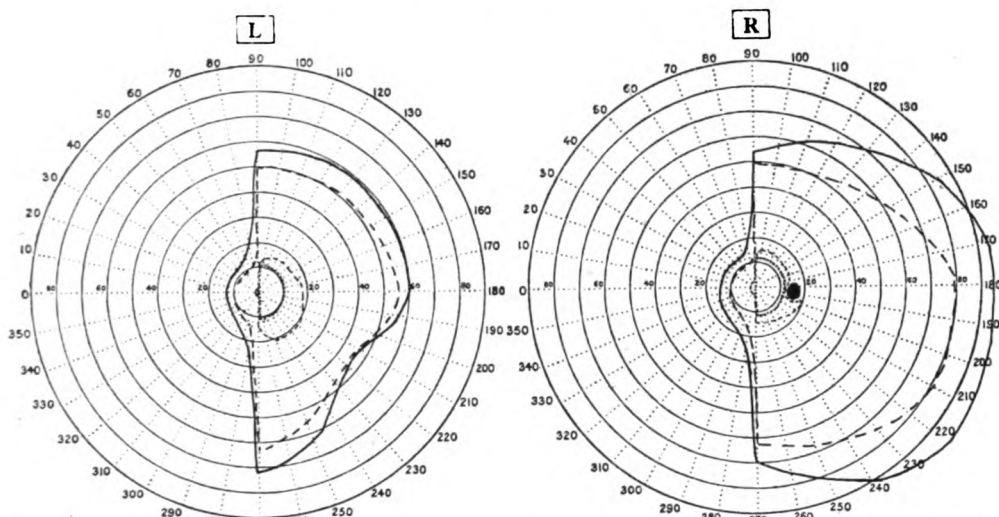


FIG. 218.—HOMONYMOUS HEMIANOPIA FROM ARTERIAL ANGIOMA NEAR RIGHT LATERAL GENICULATE BODY.

Objects R. and L. : $\frac{0.9}{3.3 \text{ o.}}, \frac{3.3 \text{ o.}}{2.1 \text{ o.}}, \frac{2.1 \text{ o.}}{3.3 \text{ o.}}$ red (dotted line).
Pronounced sparing of fixation area for white but not for red. (J. M., 1929.)

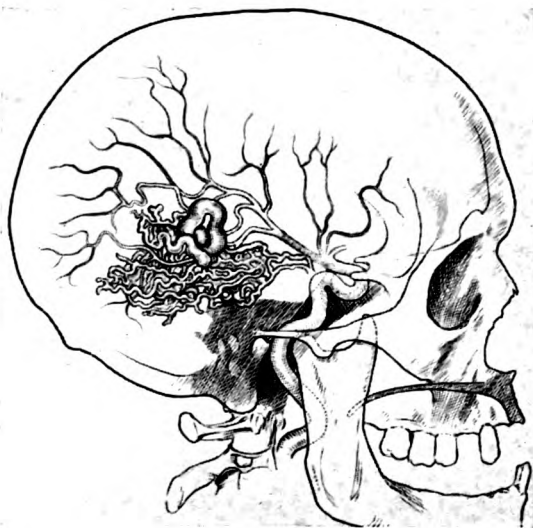


FIG. 219.—DRAWING FROM A CEREBRAL ARTERIOGRAPH SHOWING THE POSITION OF THE ANGIOMA CAUSING THE HEMIANOPIA IN FIG. 218. (SUPPLIED BY MR. NORMAN DOTT.)

bitemporal hemianopia from syphilis, tend to be more sudden in onset and associated with more rapid and greater fluctuations both in extent and intensity than occur in tumour cases. The prognosis is fairly good if the correct diagnosis is made at an early stage, and in this respect perimetry can be of some assistance. Softenings due to syphilitic or other vascular disease occur, but are not common. The defects produced appear suddenly or rapidly and remain permanent.

Injury

Homonymous defects may occur in rare cases after indirect injury.

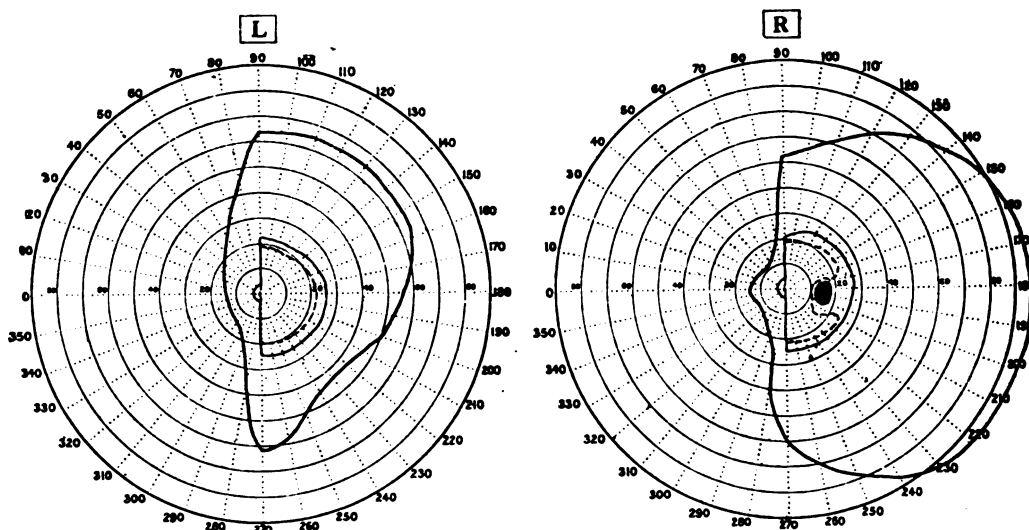


FIG. 220.—HOMONYMOUS HEMIANOPIA FROM GLIOMA OF RIGHT PARIETAL LOBE. MALE, AGE 50. Objects π_0^0 , π_0^1 , π_0^2 . Slight incongruity for large object possibly due to somewhat sloping edge of defect. Fixation area spared for π_0^0 , not for π_0^1 . Enlargement of blind spot. V.R. and L. $\frac{2}{3}$. (Ly., 13/5/23.)

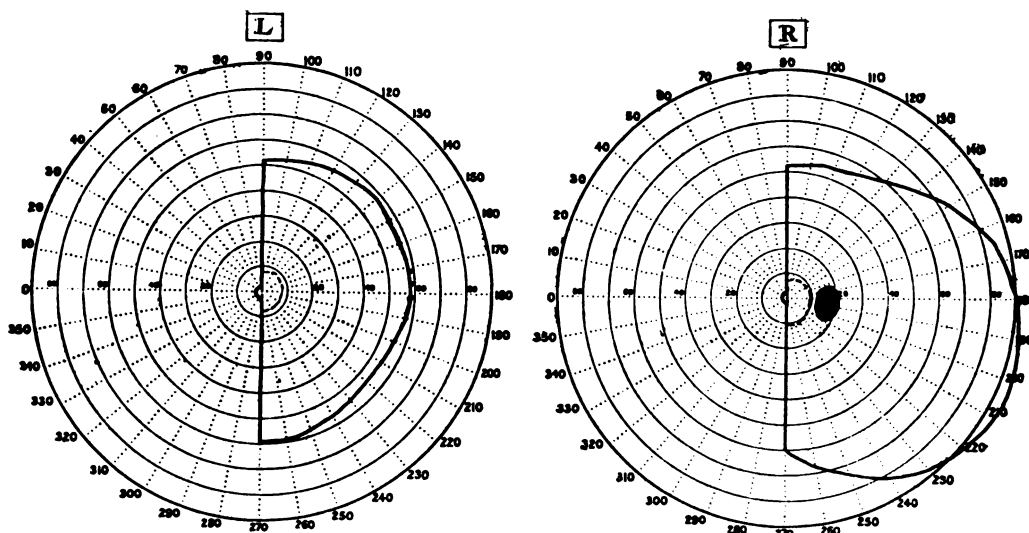


FIG. 221.—SAME FIELDS AS IN FIG. 220 A FORTNIGHT LATER. Clean cut hemianopia with sparing of fixation area for π_0^0 , not for π_0^1 . Depression of π_0^0 field. Blind spot further enlarged. R.V. : L.V. $\frac{2}{3}$. (Ly., 25/5/23.)

GENICULO-CALCARINE PATHWAY

At and above the level of the lateral geniculate body homonymous hemianopia of geniculo-calcarine type is produced by any interference with the visual path. Within the limits of the affected half-fields the defects may vary greatly in respect of position, shape, size, and intensity, but, with the exception of the temporal crescent, are always congruous and simultaneous. Any defect in one field is accompanied by another in the homonymous half-field of the other eye, simultaneous in onset, similar in position and

identical in size, shape, intensity and behaviour. In other words, a supragenulate field defect is a single defect of the binocular field. The onset may be sudden or gradual, the course mobile or stationary, and the duration temporary or permanent. These variations reflect the nature of the causal lesion, and we may regard as typical of geniculocalcarine hemianopia the field changes commonly produced by occipital vascular obstruction, sudden in onset, stationary and often complete and absolute, or showing more or less recovery, congruous and with well-marked sparing of the fixation area, in contradistinction to the gradual, progressive, uneven, incongruous subgenulate type.

The determination of the site of interference, which may be in the lateral geniculate body, the optic radiation, or the visual cortex is guided by the study of the characters of the field changes together with any other symptoms which may be present.

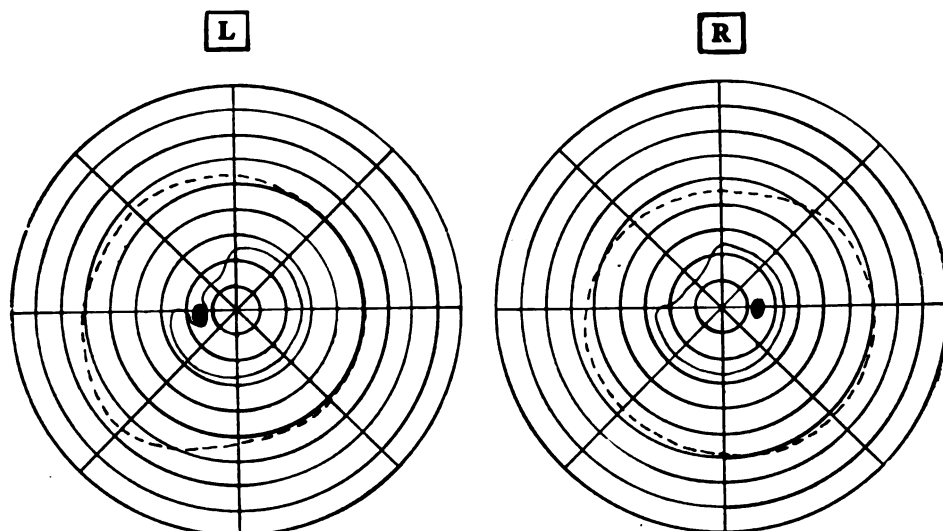


FIG. 222.—HOMONYMOUS CONGRUOUS HEMIANOPIA FROM PARIETAL TUMOUR. NOTE RESEMBLANCE TO INCIPIENT BITEMPORAL HEMIANOPIA (FIG. 154). R.V. $\frac{3}{4}$. L.V. $\frac{1}{4}$. (C., 1943.)

In the anterior part of its course the optic radiation is closely related to other nerve tracts, so that hemianopia due to lesions of this portion forms one of several symptoms of nerve interference. Behind the level of the post-central gyrus hemianopia, especially left hemianopia, may occur as an isolated symptom, and may be due to damage to the optic radiation or visual cortex. Apart from associated symptoms, there is little in the nature of the field changes which can be relied upon to distinguish hemianopia due to a lesion of the optic radiation from one depending on an affection of the cortex or to indicate the level at which the radiation is involved. It has been found by Lenz that sparing of the fixation area is less common the more anterior the lesion, an observation which indicates the greater liability of the optic radiation to complete obstruction where it is most concentrated. To a large extent, therefore, the determination of the antero-posterior level of the interference in the path depends on the presence or absence of associated symptoms. Moreover, hemianopia as an isolated symptom is so rarely due to a lesion of the optic radiation, and so frequently to one of the occipital cortex, or rather

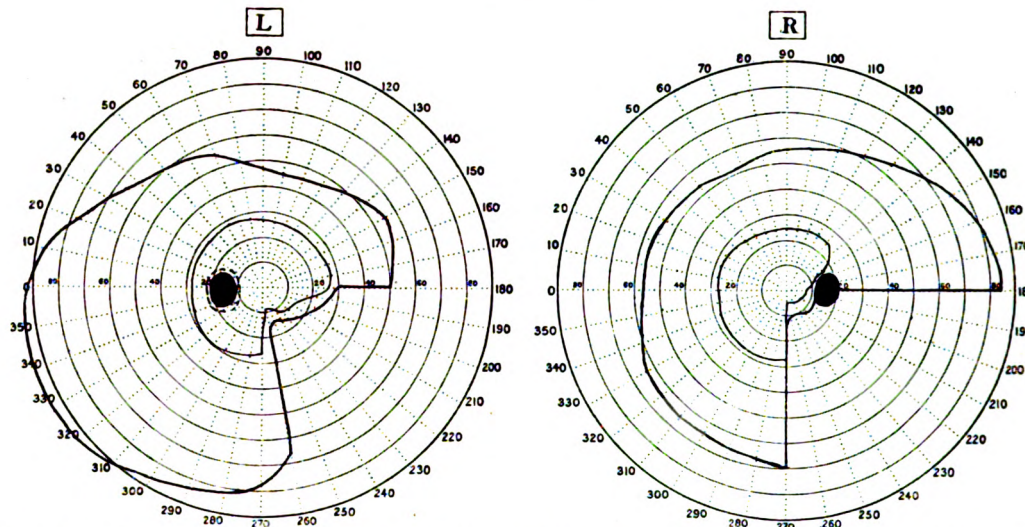


FIG. 223.—RIGHT LOWER QUADRANT HOMONYMOUS HEMIANOPIA DUE TO TUMOUR (MENINGIOMA) INVOLVING POSTERIOR TEMPORAL AND LOWER PARIETAL AREAS OF LEFT SIDE COMPRESSING THE OPTIC RADIATION.

Illustrating a large tumour producing field changes, of which the patient was quite unaware, as almost the only sign. Immediately before operation, nine days later, transient complete hemianopia developed. Objects $\frac{3}{8}$ r., $\frac{1}{8}$ l., blind spot $\frac{2}{8}$ o. V.R. and L. $\frac{5}{8}$ plus. The other signs were slight aphasia, occasional facial spasms and papilloedema. (J. S., 14/8/25.)

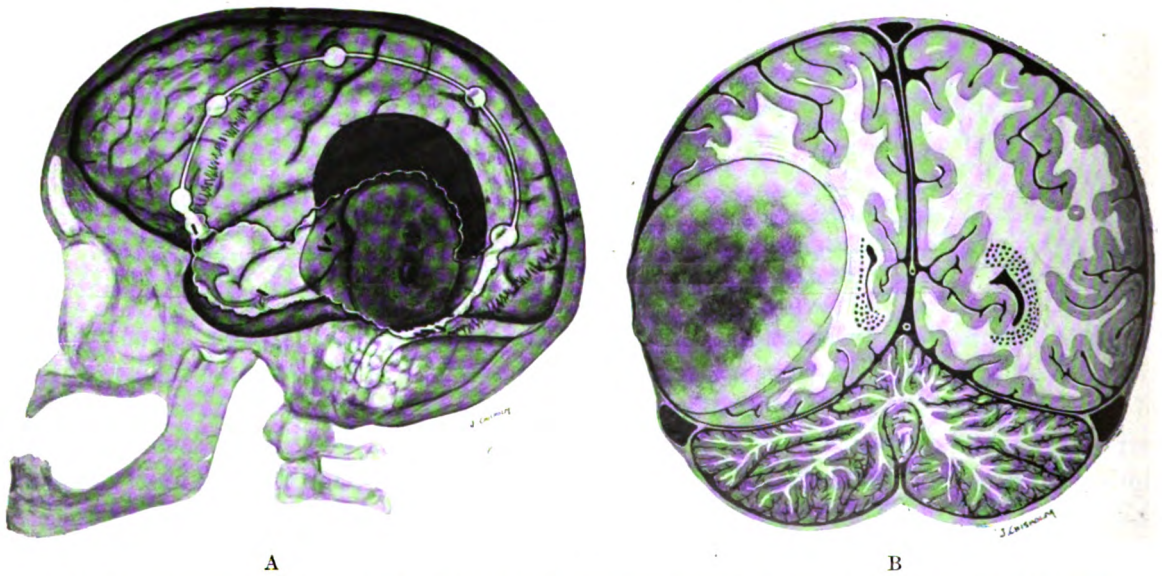


FIG. 224.—DRAWINGS SUPPLIED BY MR. NORMAN DOTT, SHOWING POSITION OF TUMOUR CAUSING FIELD CHANGES IN FIG. 223 :

A. Lateral aspect of tumour. B. Relation to optic radiation, compressing upper portion especially. (J. S., 1925.)

of the cortex plus the subcortical white matter, that the lesion may be supposed to be in the latter position unless there is some evidence, such as a wound, to indicate the former.

Roenne has, however, pointed out that quadrant hemianopia with rectilinear delimitation along the vertical and horizontal meridians of the field is to be regarded as

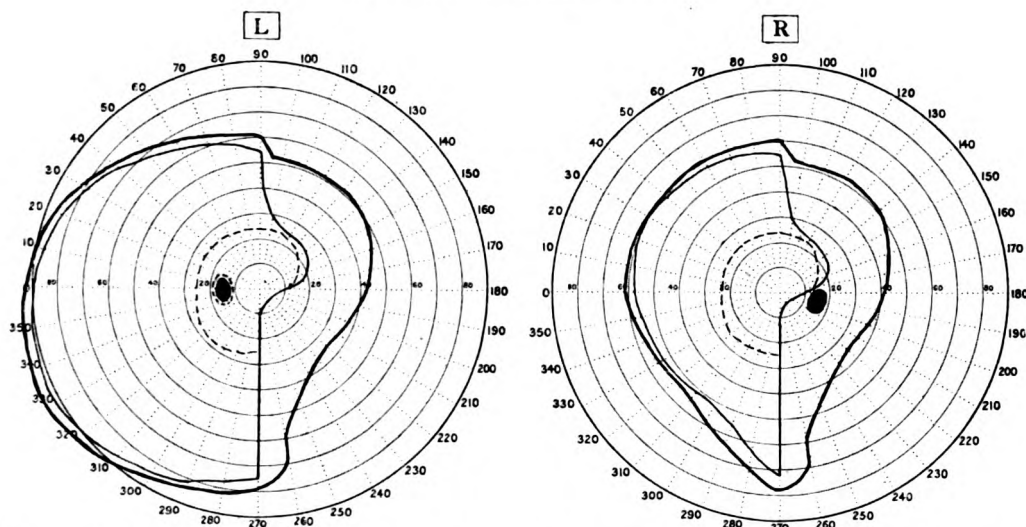


FIG. 225.—THE SAME FIELDS AS IN FIG. 223 ELEVEN WEEKS AFTER REMOVAL OF TUMOUR: RELATIVE HOMONYMOUS HEMIANOPIA.

Blind spots much smaller. Objects $\frac{5}{3} \frac{0}{3} \frac{0}{3}$, $\frac{5}{3} \frac{3}{3} \frac{0}{3}$, $\frac{1}{2} \frac{0}{0} \frac{0}{0}$.

A year later fields reported normal: V. $\frac{6}{8}$. (J. S., 25/11/25.)

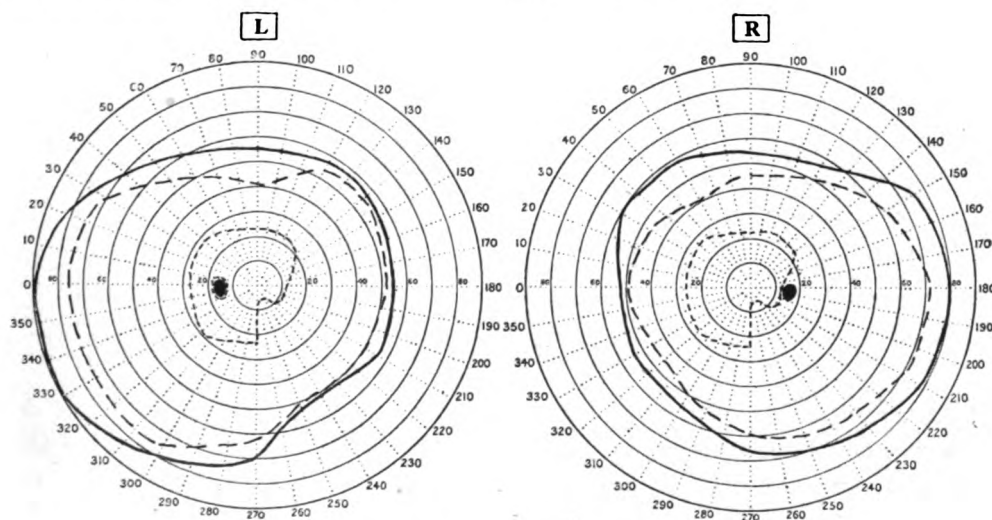


FIG. 226.—THE SAME FIELDS AS IN FIG. 225 NINE YEARS LATER.

Lower right homonymous partial quadrant hemianopia still remaining.

R.V., L.V., $\frac{6}{8}$. Objects $\frac{3}{3} \frac{3}{3} \frac{0}{0}$, $\frac{3}{3} \frac{3}{3} \frac{0}{0}$, $\frac{1}{2} \frac{0}{0} \frac{0}{0}$. The defect was more definitely quadrantal with $\frac{2}{2} \frac{0}{0} \frac{0}{0}$ red. (J. S., 1934.)

due to a lesion of the radiation, while irregularity of outline and steepness of the margins indicate cortical damage.

The vertical relationship of the lesion to the visual path is more easily determined. Since the visual fibres preserve their relative positions between the external geniculate body and the occipital cortex, a low field defect will indicate a high lesion and vice versa.

The diagnosis of the nature of the causal lesion is based upon the onset, course and intensity of the field defects. The most common causes are vascular changes, tumours

and abscesses which act either by interfering with the nutrition of the nerve elements to a greater or less extent, or by destroying them. In the former case function is impaired, but not permanently abrogated; in the latter absolute and permanent functional loss ensues. When the onset is sudden the causation is usually vascular, and may be due to thrombosis, hæmorrhage or embolism. Frequently the defect is severe at first (there may be initial blindness) and improves to a certain extent, the permanent field change representing the amount of destruction. Sudden relative hemianopia may be ascribed to partial impairment of nutrition by hæmorrhage or œdema, pressing upon but not destroying the visual fibres as in the transient hemianopia which may accompany hemiplegia. Transient hemianopia may also be produced by angiospasm, which is also

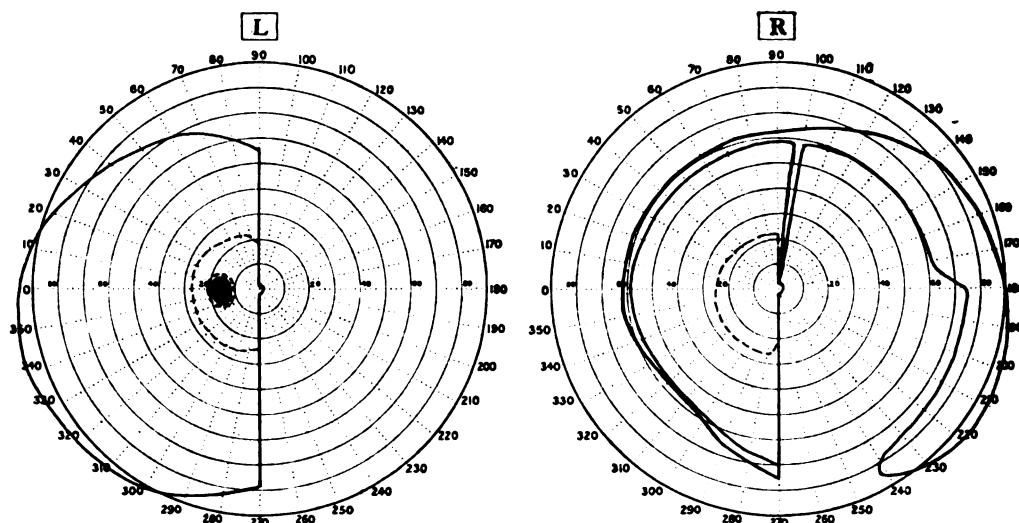


FIG. 227.—HOMONYMOUS HEMIANOPIA WITH TEMPORAL CRESCENT OR "HALF MOON."

Tumour involving optic radiation. No symptoms other than headache, choked discs and hemianopia. Objects $\pi\frac{1}{2}0$, $\pi\frac{1}{2}0$; $\pi\frac{1}{2}00$. In the right field temporal crescent or "half moon" found with $\pi\frac{1}{2}0$, not with $\pi\frac{1}{2}0$. R.V. $\frac{1}{2}$. L.V. $\frac{1}{2}$. The fibres of the optic radiation corresponding to the unpaired area of the field have been partially spared. The obliquity of the upper part of the dividing line in the right field was not explained. (Rh., 1924.)

regarded as the cause of recurrent temporary hemianopia in migraine. It is doubtful whether permanent homonymous hemianopia is ever a sequel of true migraine though it may, of course, be caused by a lesion which also produces symptoms which resemble those of migraine.

When the onset is gradual and the course progressive, tumour or abscess is indicated and the position, extent, intensity and course of the field changes reflect the same features in the lesion. As the brain accommodates itself to some extent to distortion from a tumour, especially if slow-growing, the swelling may reach a considerable size, depending on its situation, before field changes are produced which for this reason may be a relatively late symptom when the visual path is not primarily involved. Owing to the gradual onset the patient is often unaware of the commencement of the defect whereas he usually notices immediately the hemianopia caused by a vascular lesion. The more carefully the fields are tested with small visual angles in the early stages of, suspected cases, the sooner will the presence of field changes be detected.

Lateral Geniculate Body

Isolated lesions confined to this nucleus are extremely rare, and as far as is known no distinctive perimetric signs are produced. Syphilis, softenings, hæmorrhages and tumours affecting the neighbouring structures may involve this part of the nerve path directly or indirectly. The upper part of the nucleus, being nearer the common site of hæmorrhages, is more exposed to permanent damage. Both lateral geniculate bodies may be affected at the same time by syphilitic arterial degeneration (Mackenzie 274).

Incongruous homonymous hemianopia is produced corresponding to the extent of the interference with conduction. The defect may be complete at first, resolving later into an inferior quadrant hemianopia as the lower portion of the visual path becomes

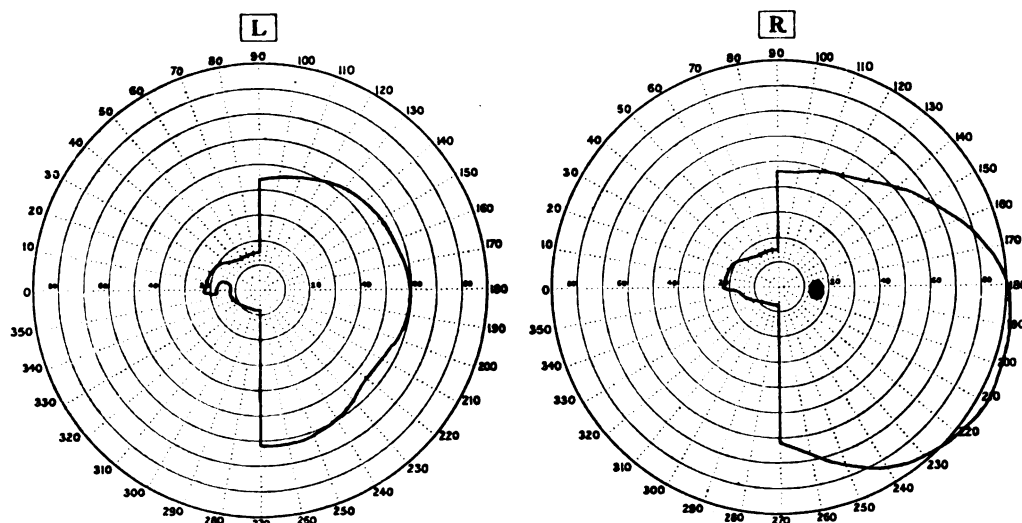


FIG. 228.—HOMONYMOUS HEMIANOPIA FROM VASCULAR DISEASE.

Duration one day; sudden onset; no other symptoms. Male, age 64. V. R. and L. $\frac{1}{2}$. Unusually large spared area in central region. Object $\frac{3}{8}$ 0. (Mn., 18/6/22.)

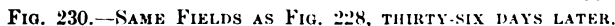
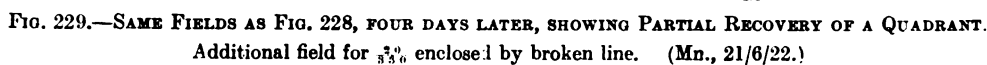
relieved from pressure. Since the termination of the tract or the beginning of the optic radiation may also be affected, it is not possible to diagnose interference with the geniculate body alone during life, though a lesion of the visual path in or near the posterior part of the internal capsule may be indicated (Malbran 277).

Optic Radiation and Occipital Visual Cortex

Lesions of the internal capsule, especially if deep, may produce hemianopia with associated symptoms of sensori-motor interference.

Affections of the parietal lobe, if low enough, may cause homonymous defects varying from impairment of the inferior quadrants to a complete hemianopia. Relative homonymous hemianopia affecting the fixation area sufficiently to interfere with reading and existing for six months before the appearance of other symptoms has been recorded by Throckmorton in a case of tumour of the parietal lobe, and other similar cases have been observed.

Swellings of the temporal lobe such as tumour or abscess involve the visual path



The temporal crescent in the left field has recovered causing the quadrant defect to appear as a quadrant scotoma. In the right nasal field it appears as a complete quadrant loss since there is no peripheral area corresponding to the temporal crescent. Objects $\frac{3}{4} \frac{3}{4} \frac{3}{4}$, $\frac{3}{4} \frac{3}{4} \frac{3}{4}$. Scotoma $\frac{3}{4} \frac{3}{4} \frac{3}{4}$. (Mn., 30 7 22.)

by pressure on the tract or, if sufficiently deep and extensive, on the lateral geniculate body or the optic radiation at or near its beginning. Homonymous field defects are very common and of great value in diagnosis. Tract interference from this cause has already been referred to and would appear to be the usual form of visual interference. Supragenicular hemianopia due to a lesion in this area presents no special perimetric features.

Posterior to the parietal region the optic radiation is not frequently affected **without** associated involvement of the cortex except by hæmorrhage, and in some cases **of injury**

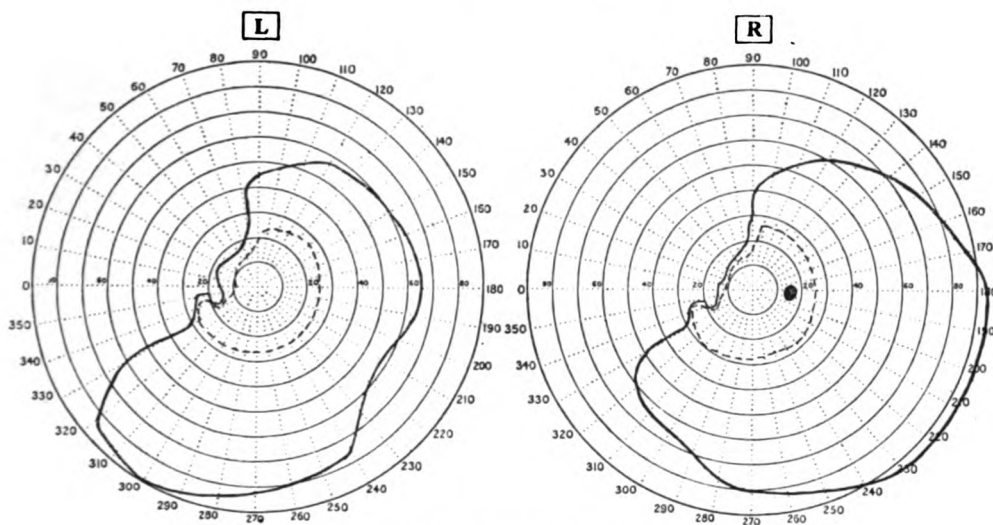


FIG. 231.—PARTIAL HOMONYMOUS HEMIANOPIA.

Patient aged 26. A week previously left hemiparesis lasting ten minutes followed by hemianopia which remained.
 Diagnosis: Arterial spasm (?). In good health May, 1937.
 R.V. $\frac{6}{8}$. L.V. $\frac{6}{24}$. Objects $\frac{3}{300}$, $\frac{5}{1000}$. (G. R., 1927.)

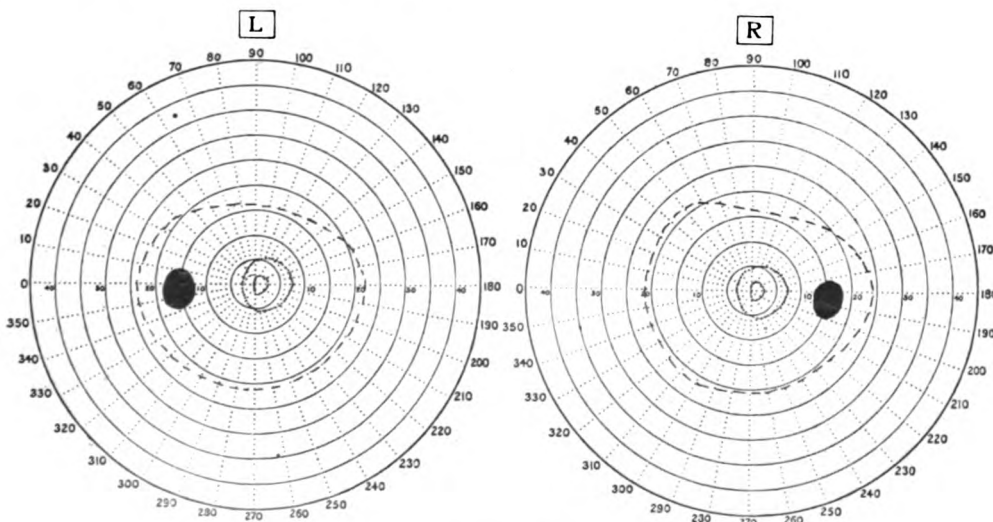


FIG. 232.—HOMONYMOUS HEMIANOPIA.

To show remains of field defect after apparent recovery. History of hemianopia 18 months previously.
 Objects: $\frac{2}{2000}$ white; $\frac{2}{2000}$ red (dotted line).

Field for white normal but colour fields show homonymous defects. Peripheral fields normal.

R.V. $\frac{6}{8}$. L.V. $\frac{6}{8}$. (Ln., 1929.)

and in the early stages of tumour. In radiation interference due to tumour the hemianopia begins slowly and gradually and is at first of very low intensity. The onset may not be noticed by the patient.

In the occipital lobe both the cortex and the subjacent white matter are commonly involved together unless the lesion is minute. Frequently the lesion is extensive and the hemianopia complete with sparing of the fixation area. The usual form is that due to

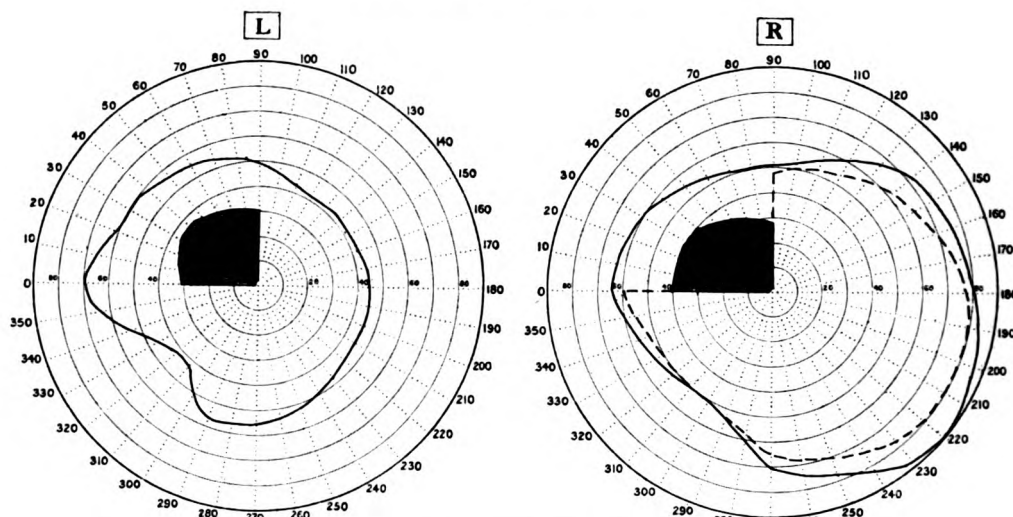


FIG. 233.—HEMIANOPIC QUADRANT SCOTOMA IN VASCULAR DISEASE. FEMALE, AGE 70.

Fixation area spared on each side. Objects $\frac{5}{32}$ 0, $\frac{2}{32}$ 0. There was nuclear cataract in the left eye, which caused the field to appear contracted. R.V. $\frac{5}{32}$. L.V. counting fingers at 1 m. (Sd., 1911.)

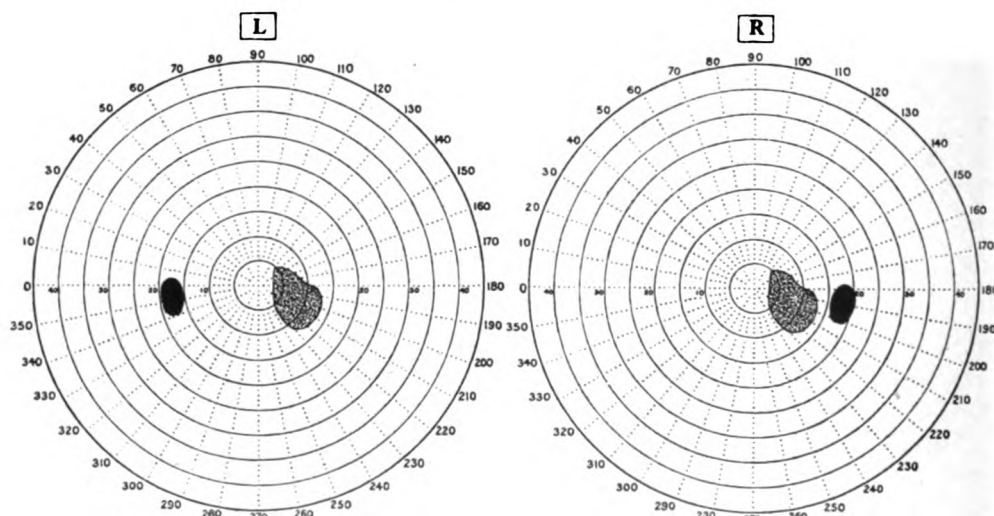


FIG. 234.—CONGRUOUS HOMONYMOUS PARACENTRAL HEMIANOPIC SCOTOMATA OF SUDDEN ONSET.

The defects were absolute and steep-edged and probably cortical in origin. The only symptom was difficulty in reading. $\frac{5}{32}$ 0. R.V., L.V. $\frac{5}{32}$. (J. S., 1930.)

occlusion of the calcarine artery and occurs suddenly or rapidly. It may occur during sleep or at any time. General symptoms are frequently associated and, when the patient is well enough to have the fields examined, complete and absolute hemianopia with central sparing will usually be found. In other cases the hemianopia is incomplete or relative or both. Recovery takes place slowly and traces may persist in the central field after a year (Fig. 232). Transient hemianopia may be due to arterial spasm.

In incomplete examples due to cortical damage the defects are more peripheral

when the lesion is in the anterior part of the cortical visual area, and more central the nearer it is to the occipital pole. Homonymous quadrant defects are referable to damage to the upper or lower lip of the calcarine sulcus or to the upper or lower segment of the radiation. Only a narrow sector in each field may be affected or a quadrant or rather more, the defects differing from complete hemianopia only in extent. Sometimes the unpaired portion of the temporal field is intact or only relatively impaired, indicating that the lesion does not involve the most medial fibres of the radiation or, if cortical, does not extend to the anterior limit of the visual area. Holmes (187) has pointed out that the remarkably clean cut straight horizontal margin of such defects in injuries of

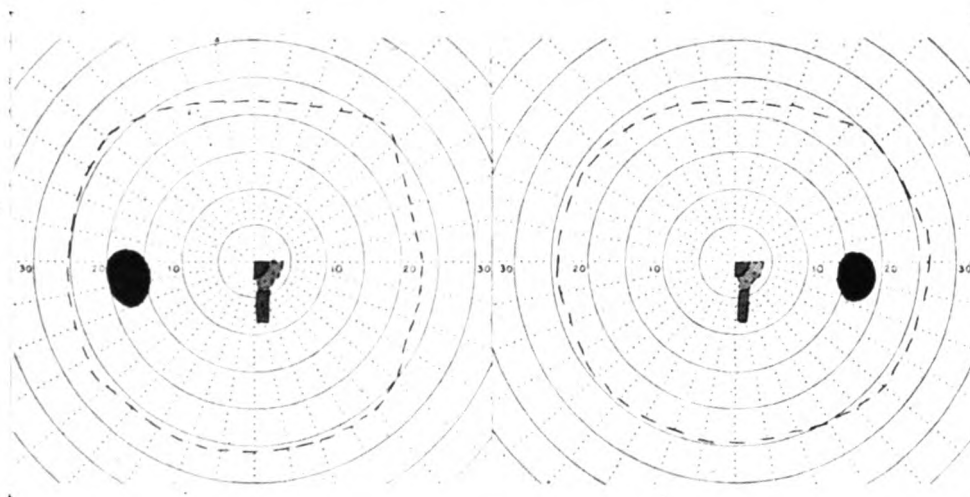


FIG. 235. CENTRAL HOMONYMOUS HEMIANOPIC QUADRANTIC SCOTOMATA REFERABLE TO MINUTE LESION IN LEFT MACULAR CORTX.

V.R. and L. $\frac{1}{2}$ part. Field for $\frac{1}{2}$ normal in extent. Scotomata almost absolute. (He., 1923.)

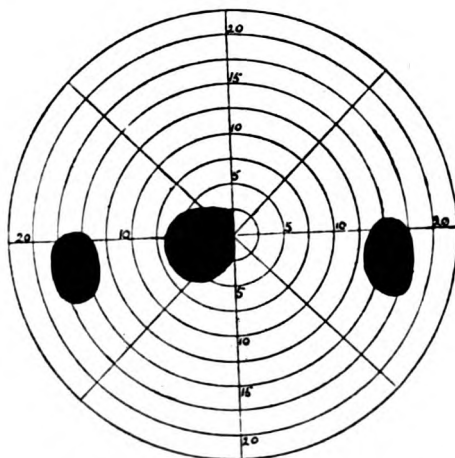


FIG. 236 CENTRAL HOMONYMOUS HEMIANOPIC CONGRUOUS SCOTOMATA. FEMALE, AGE 34. RIGHT AND LEFT FIELDS SHOWN COMBINED.

Following a quarrel in which she was stabbed in the shoulder, the patient found difficulty in reading. Scotomata are absolute, steep-edged and exactly congruous. R.V. $\frac{1}{2}$. L.V. $\frac{1}{2}$. (H., 1941.)

the radiation indicates the possibility that the fibres corresponding to the upper and lower retinal quadrants may be separated by an anatomical interval, a view supported by Roenne (344, 350), who thinks that such an interval exists and is occupied by the macular fibres. In the case of the cortex there is no anatomical reason why a quadrant defect should have a clean cut straight horizontal boundary and homonymous hemianopic quadrant defects with irregular outlines and steep edges may therefore be regarded as of cortical origin. There is also evidence that the field along the vertical meridian corresponds to the superficial part of the visual cortex, while the part along the horizontal meridian corresponds to the bottom of the calcarine sulcus.

Though no form of field defect is pathognomonic of a cortical lesion the presence of small central homonymous hemianopic scotomata is very suggestive of cortical or sub-

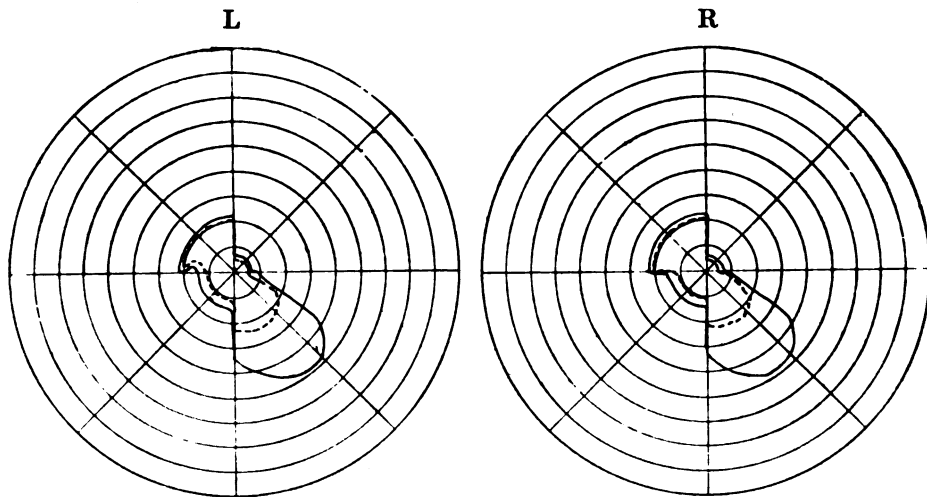


FIG. 237.—BILATERAL HOMONYMOUS CONGRUOUS HEMIANOPIA.

On the right side the upper quadrant is affected and the upper part of the lower quadrant. On the left side the lower quadrant. In addition there is general depression of the fields. Vision R. and L. $\frac{6}{60}$. Object $\frac{3}{36}$ and $\frac{3}{60}$. The optic discs were healthy in appearance. The field defects were found ten weeks after an acute attack of ependymomeningitis, probably meningococcal. Damage to the walls of the lateral ventricles ascertained by ventriculography. Impairment of memory present, but dysphasia and other signs of cortical interference absent. (A. M., 1941.) (With acknowledgments to Mr. Norman Dott.)

cortical damage if they occur suddenly in otherwise normal fields and without other symptoms. They may occur spontaneously or after injury, and usually affect the apex of one quadrant only. They are usually isolated, but may be associated with areas of less intense defect, and the fixation area is involved in most cases though often only by a narrow tongue. Central vision is nevertheless usually good as only part of the fixation area is affected, but reading or figuring may be impaired according to the position of the scotoma. The fixation area is sometimes, but rarely, spared. Small homonymous paracentral scotomata (Fig. 234), in which the symptoms are similar but the area along the vertical meridian is unaffected, are also found. In such cases the question of sparing of the fixation area does not arise. Very few of these defects have been exhaustively examined by quantitative perimetry. More peripherally situated homonymous scotomata presumably occur, but are unlikely to be detected as they do not attract

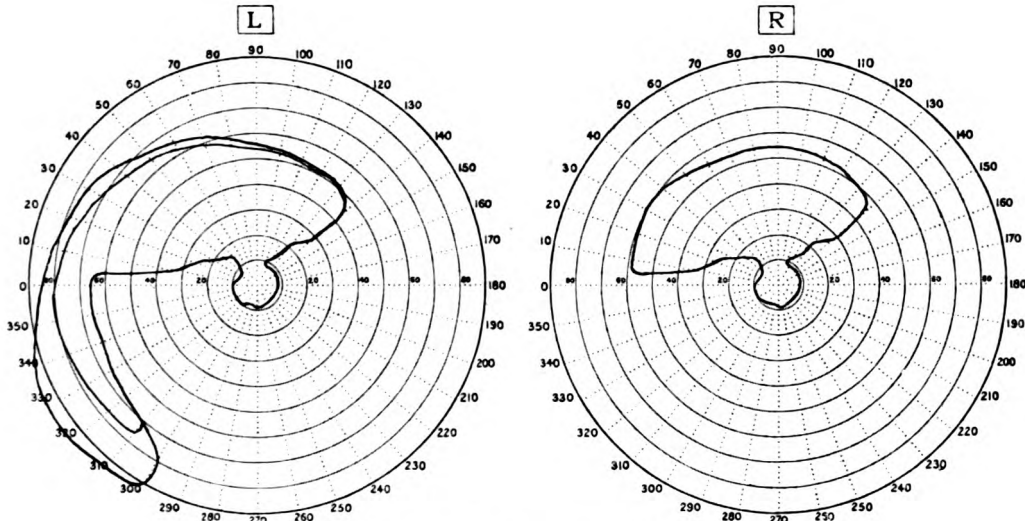


FIG. 238.—BILATERAL HOMONYMOUS HEMIANOPIA.

Objects: R. $\frac{6}{330}$. L. $\frac{6}{330}$, $\frac{5}{330}$. V.R. and L. $\frac{6}{330}$.

Note symmetry of fields with the exception of the temporal crescent on left side. Evidently the upper lip of the calcarine sulcus on each side is mainly affected, but the anterior part of the visual cortex on the right side has been spared leading to the preservation of the temporal crescent in the field of the left eye. Onset apparently simultaneous on the two sides. (R. L., 1929.)

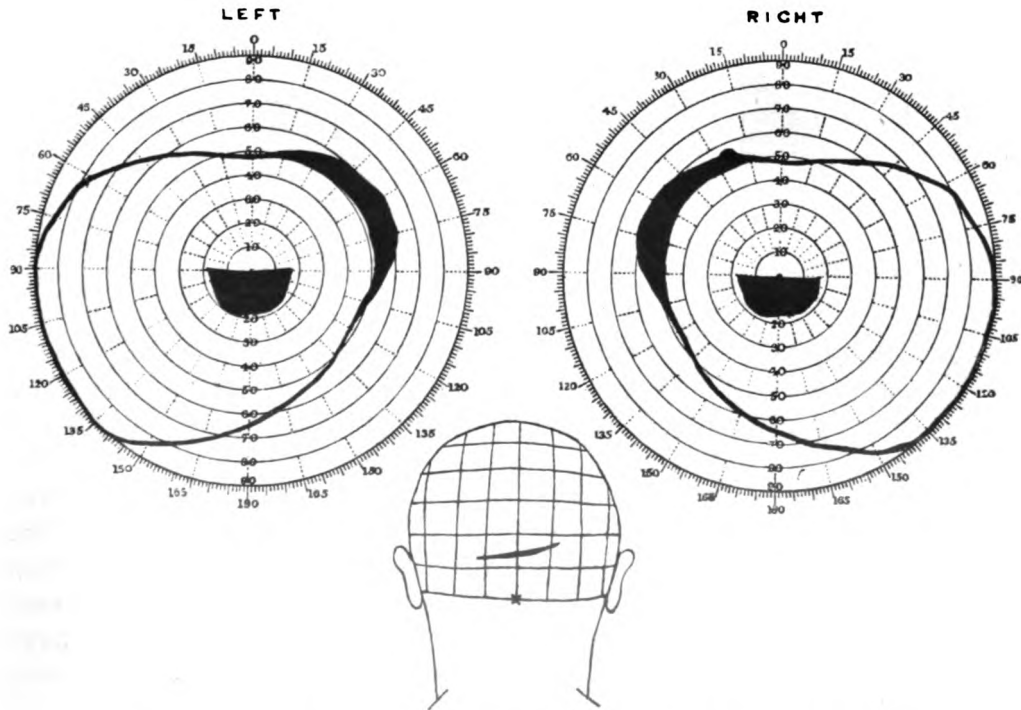


FIG. 239.—OCCIPITAL INJURY (SHELL FRAGMENT). BILATERAL INFERIOR HEMIANOPIC QUADRANT SCOTOMATA. GORDON HOLMES.

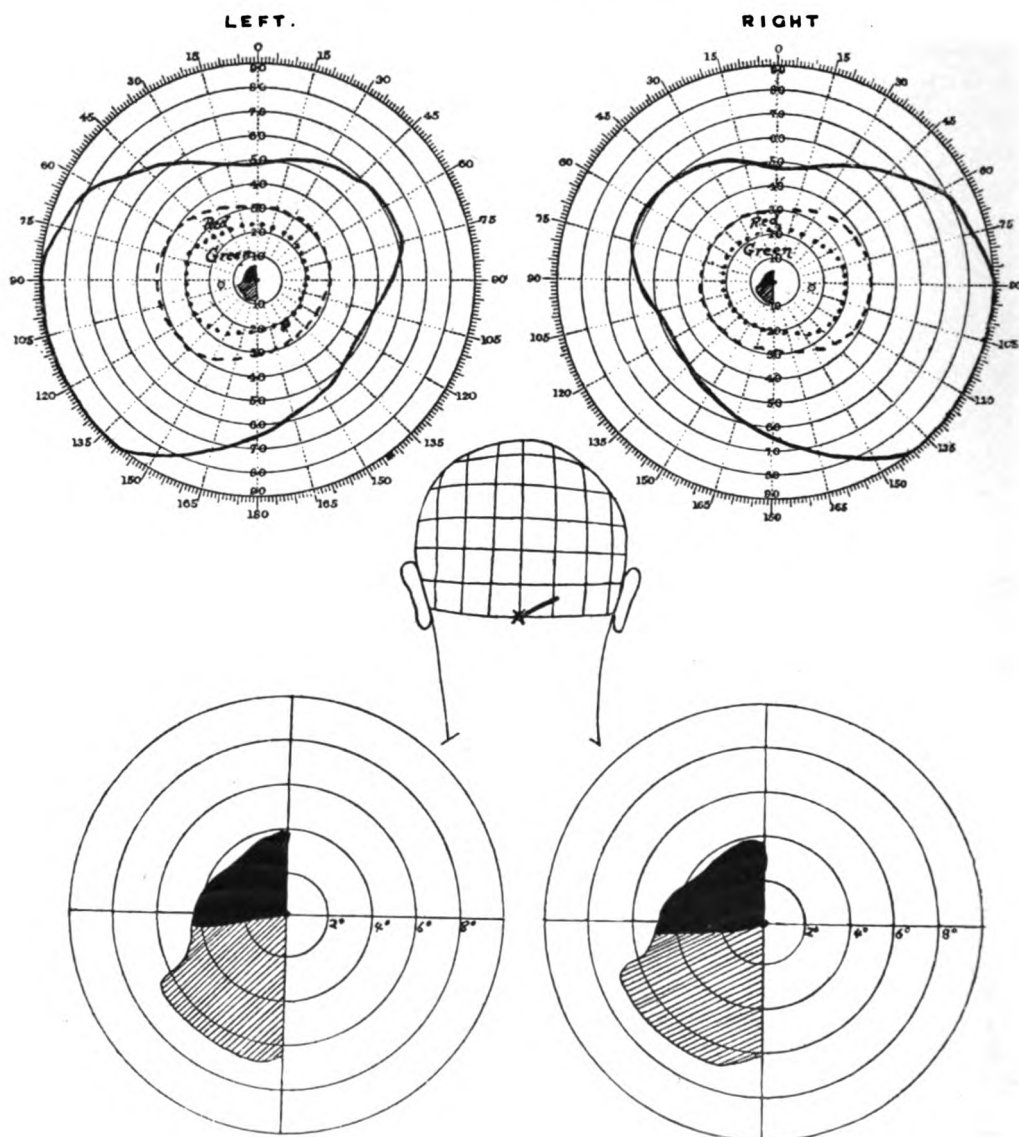


FIG. 240.—INJURY TO TIP OF RIGHT OCCIPITAL LOBE. LEFT HEMIANOPIC CENTRAL QUADRANT SCOTOMATA, EACH ANALYSED INTO TWO QUADRANT SCOTOMATA OF DIFFERENT INTENSITIES. GORDON HOLMES.

attention. While such defects may theoretically be caused by a lesion anywhere in the suprachiasmal part of the pathway the history, character and course suggest that the cause is vascular and the site cortical or sub-cortical, for example, obstruction of a small vessel in the tip of the occipital lobe after it has left the pial anastomosis and pierced the cortex. Some cases are due to injury (Figs 240, 244) and others have been reported in uræmia (Salus, 359). As the suprachiasmal path is more spread out at this part than at any other minute isolated non-progressive defects of this type are more likely to be produced by lesions here than at a lower level.

Apart from these scotomata, sparing of the fixation area is almost constant in

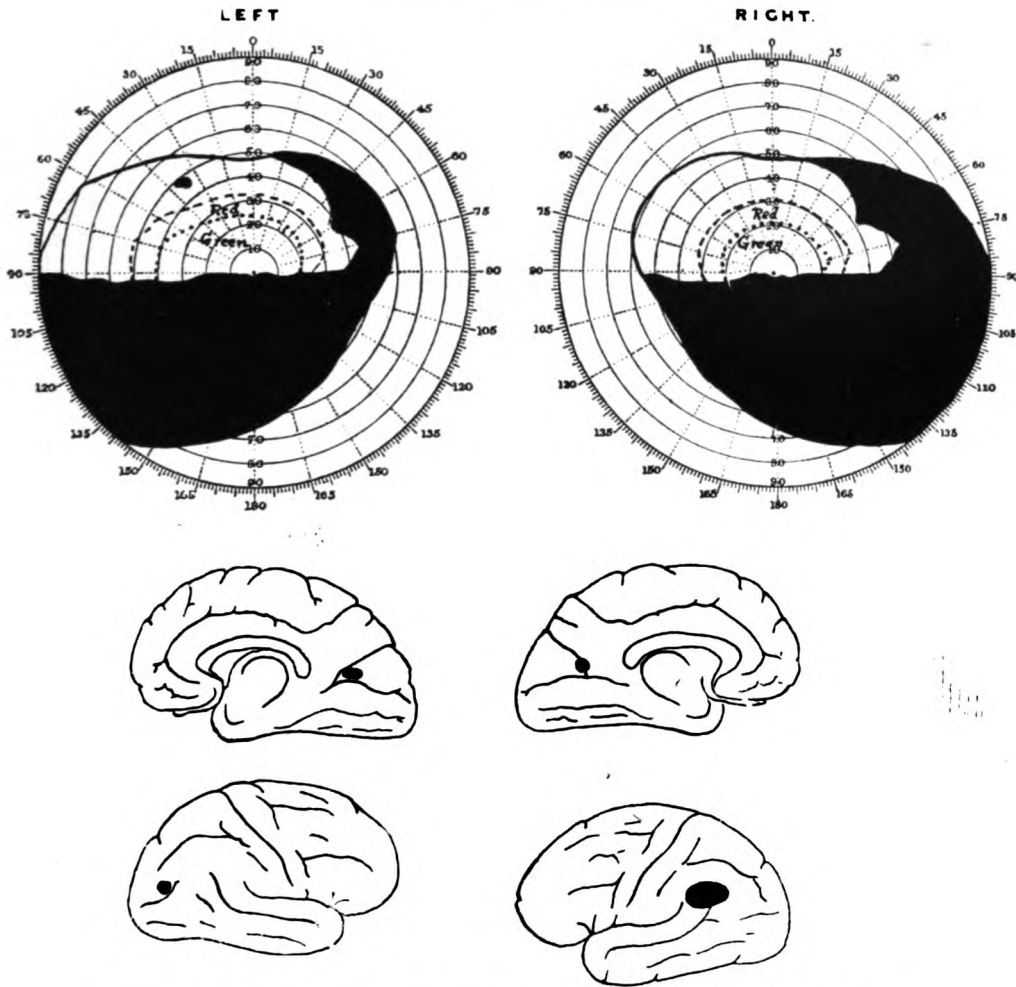


FIG. 241.—DESTRUCTION OF SUPERIOR PORTIONS OF OPTIC RADIATIONS OF BOTH SIDES.
V.R. AND L. $\frac{1}{2}$. GORDON HOLMES.

occipital hemianopias of vascular origin, and is more extensive and more complete than in subgeniulate hemianopia. The spared area is usually less than 5° in extent but may be more, and has been found occasionally to extend upwards or downwards or in both directions along the vertical meridian for some distance merging into the overshoot field, which attains its fullest expression in exceptional cases where the sparing extends to the periphery both above and below. The boundary for colour conforms nearly always to that for white. Sometimes the sparing is not present at first, but appears in a few days. The most probable explanation is the double vascular supply of the tip of the occipital lobe, but the possibility of an error of observation should not be forgotten and the exact conditions should be worked out on the screen.

Bilateral homonymous hemianopia is usually due to lesions of the cortex or radiations produced by vascular disease or injury. It may also be produced by ependymitis of the lateral ventricles (Fig. 237). In cases due to hæmorrhage or obstruction the

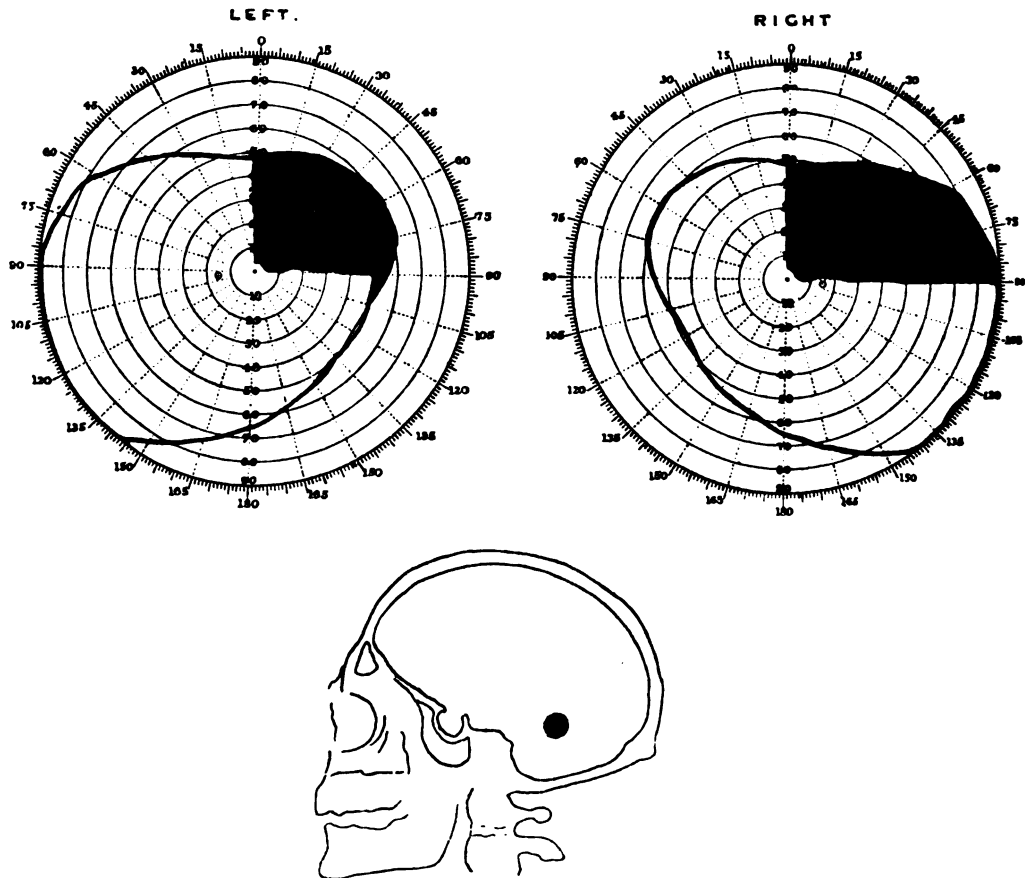


FIG. 242.—INJURY TO LOWER PORTION OF LEFT OPTIC RADIATION. V.R. AND L. f.
GORDON HOLMES

two sides may be affected in sequence, or, rarely, simultaneously. Frequently only a minute central field remains corresponding to the bilateral sparing of the fixation area. In about a quarter of the cases (Lenz) complete blindness remains. In others the most varied field pictures result, as all degrees of incomplete or relative defect may be produced on the two sides in different combinations (Fig. 238).

These differences when present sometimes enable fields of this kind to be split up by analysis into two hemianopias showing their true nature even when only very small central fields are present.

Bilateral homonymous hemianopic central scotomata without peripheral changes have not been recorded except in connection with injury of both occipital lobes alone. In such cases various combinations of bilateral homonymous quadrantic central scotomata with intact peripheral fields have been found (Fig. 239).

War injuries have proved a fruitful source of bilateral hemianopias, and much of our knowledge of the occipital visual centre has been derived from the study of the fields in occipital wounds. The relation of the field defects to the anatomical position of the injury is very exact. Transverse bullet wounds frequently produce horizontal hemia-

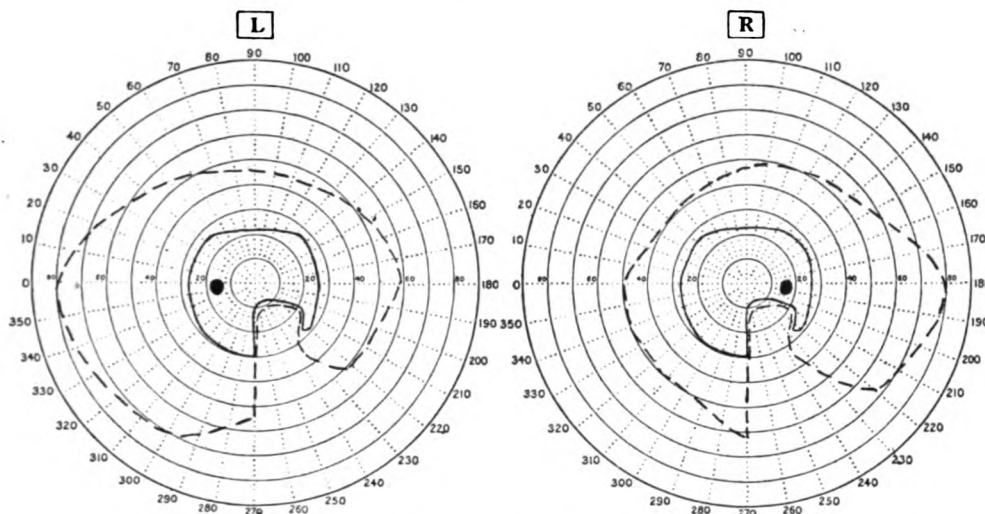


FIG. 243.—PARTIAL HOMONYMOUS LOWER QUADRANT HEMIANOPIA FROM INJURY.

In 1918 gunshot wound of left upper occipital region close to middle line. Subsequent attacks of hemianopic hallucinations and headache. Diagnosis: Cortical cicatrization. R.V., L.V., $\frac{4}{4}$. Objects $\frac{1}{3} \frac{4}{0} \frac{0}{0}$. (W. S., 1931.)

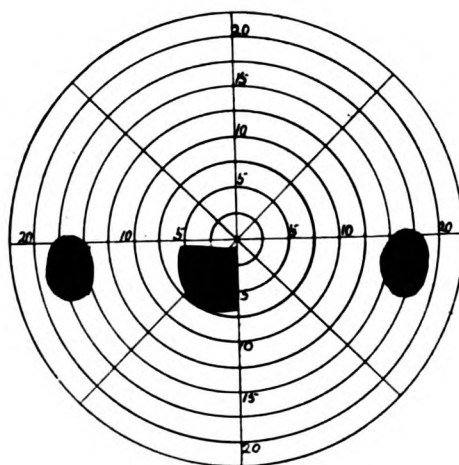


FIG. 244.—LEFT LOWER HOMONYMOUS CONGRUOUS ABSOLUTE HEMIANOPIC SCOTOMA FROM OCCIPITAL GUNSHOT WOUND IN 1917. (BINOCULAR.)
R.V. $\frac{4}{4}$. L.V. $\frac{4}{4}$. Objects $\frac{4}{0} \frac{0}{0} \frac{0}{0}$. (G., 1942.)

nia, due to damage to the visual area or its fibres on each side, a bilateral quadrant hemianopia (Fig. 241). The lower fields are nearly always affected as transverse bullet wounds involving the lower part of the visual area are usually fatal owing to the proximity of the large blood sinuses. Oblique transverse wounds produce field changes similarly corresponding to the parts of the visual area destroyed.

Homonymous hemianopia, whether uni- or bilateral, resulting from injury to the occipital lobes is much more commonly associated with involvement of the fixation area than when it is due to vascular disease.

The usual cause of occipital hemianopia is softening due to arterial obstruction. As in migraine the obstruction may be temporary and due to angiospasm. Hæmorrhage is less common here than more anteriorly. When complete blindness resolving into homonymous hemianopia occurs hæmorrhage is suggested, the initial disturbance having affected the opposite visual centre by pressure. Sparing of the fixation area developing after initial division of the central area also indicates a vascular lesion, whereas gradually arising division of the central area in a hemianopia which originally showed sparing suggests tumour.

A tumour in the occipital lobe may produce hemianopia as an isolated symptom. Growths may affect both visual centres by extension or pressure, so that blindness developing out of hemianopia suggests this form of lesion.

In hemianopia due to occipital lobe tumours, according to Horrax and Putnam (190), sparing is three times as frequent as division of the central area. Homonymous lower quadrant defects are common and upper quadrant defects rare. The defects are always congruous and the area of the field which they occupy is of variable extent. In tumour of the temporal lobe, on the other hand, upper quadrant defects are the more common, they are incongruous and tend to be right-angled. Johnson (214) found that hemianopia due to an occipital, or a frontal, lobe tumour was more likely to be complete than incomplete, and that temporal lobe tumours cause quadrant hemianopia much more frequently than occipital lobe tumours.

Homonymous hemianopia was found by Uhthoff in 9 per cent. of abscesses of the cerebrum, more than half of which were in the occipital lobe.

The prognosis as to recovery is improved if it is found that field areas previously blind begin to obtain vision for moving and large objects. Recovery commences in a part of the field which corresponds to the periphery of the lesion and colour vision is the last to be restored.

LESIONS AFFECTING THE VISUAL PATH
SCHEME SHOWING THE MORE IMPORTANT RELATIONSHIPS.

	Lesion.	Field.
Extracranial	Optic nerve	Central scotoma. Total blindness or segmental defect. Segmental defect; central scotoma. Enlarged blind spot.
		Concentric contraction.
	Subchiasmal	Uni- or bilateral central scotoma with or without peripheral defects. Unilateral hemianopic or quadrantic defects. Central scotoma on side of lesion.
		Quadrantic temporal central scotoma (junction scotoma). Unilateral temporal hemianopia leading to bitemporal hemianopia or blindness of one eye with contralateral temporal hemianopia.
Intracranial	Subgeniculate	Bitemporal hemianopia.
		Unilateral hemianopic defect leading to homonymous hemianopia.
	Chiasmal	Incongruous homonymous hemianopic quadrantic defects as a rule leading to complete hemianopia. Sparing of central area uncommon in later stages, common in early stage.
		Congruous homonymous hemianopic defects.
	Suprachiasmal	Sparing of central area common. Upper quadrantic defects rare. Peripheral defects due to medial lesion. Central defects due to lateral lesions.
		Congruous homonymous hemianopic defects. Sparing of central area common. Upper quadrantic defects rare. Homonymous hemianopic central scotomata. Peripheral defects due to anterior lesions. Central defects due to posterior lesions.
	Optic radiation	Thrombosis. Hemorrhage. Tumour or abscess. Injury.
	Occipital cortex	Thrombosis. Tumour. Injury.

CHAPTER XII

FUNCTIONAL CHANGES IN THE FIELD OF VISION

FIELD changes which are *functional* are those which are caused not by organic lesions of the oculo-calcarine visual path, but by disordered or inefficient action of parts of the brain at a much higher level. The interference is with the will of the patient, and such functions as perception, appreciation, attention, and response are involved ; in other words, it is the patient who is at fault, and not his visual mechanism. The characters of field changes produced by this kind of interference will therefore reflect the features of a disordered psyche, and not those of the anatomical structure of the visual path or of the organic lesions which may affect it.

Functional field changes may be divided into those which are manifestations of what is known as neurasthenia and those associated with what is called hysteria, terms which *faute de mieux* for practical purposes satisfactorily indicate two sufficiently characteristic symptom groups. While pronounced examples are typical of one condition or the other, doubtful or border-line cases are common, and hysterics are often neurasthenic, while neurasthenics are always suggestible.

In whatever way produced, the visual symptoms, though presenting more or less distinctive features, exhibit peculiarities, inconsistencies, and incompatibilities of a broadly similar nature which only require to be discovered to be recognised. In addition, there may be an element of wilful simulation on the part of the subject, by which field changes of similar character may be imitated, and the possibility of the coexistence of an organic lesion of the visual path should not be forgotten.

These functional field changes depend upon suggestion, and this in its turn may be due to some injury, slight or severe, or to the results of illness or overstrain, or to some mental impression. The manner in which a perimetric examination is carried out, the method used, and the personality and attitude of the perimetrist are factors of great importance. If the examination is undertaken in order to demonstrate a certain field change the quest is not unlikely to be successful. This aspect of perimetry has been clearly indicated by Roenne, who writes : " It is a peculiarity of perimetry that its outward technique is so simple as apparently to make no demand, but to be feasible for the least experienced assistant of a clinic, and yet of all ophthalmic examinations it is perhaps the one which in reality requires the most experience and detachment."

The perimetrist must therefore be thoroughly alive to the possibility of producing functional defects either alone or, what is more serious as more apt to mislead, as modifications of existing organic defects.

The most common functional field defects are general depression and concentric contraction, reversal of the colour fields (the blue field lying within the red) or interlacing of their boundaries, and some forms of ring scotoma.

An interesting manifestation, which occasionally develops when colour tests are being used, is the naming of a colour by its complementary when it is dimly or doubt-

fully seen. Thus red is called red in field areas where it is well seen ; in adjoining areas where the red colour is doubtful the response of green may be given by the patient. This phenomenon may also arise where there is an organic lesion such as tabes or tobacco amblyopia, and is probably due merely to over-anxiety on the part of the patient to reply correctly.

Hemianopic defects are rare, but may be found in patients who have in some way learned about such conditions : scotomata other than ring scotomata have only exceptionally been reported. Central scotoma is never simulated by an hysteric. I have produced a blind spot correct as to size, shape and distance from the fixation point on the nasal side of the field in a patient in whom I had immediately beforehand demonstrated the true blind spot on the other side of the same field. The suggested defect disappeared very rapidly.

These changes occur alike, though with some modifications, in neurasthenic and hysterical conditions, and no hard and fast line between the two can be drawn. When perimetric findings suggest that a functional element is present, repeated tests should be made. Care must be taken not to mistake the effects of mere nervousness, anxiety, or misunderstanding of the nature and object of the examination for pathological field changes. A little time to enable the patient to feel at ease and gain confidence often enlarges fields at first apparently contracted.

Slight or moderate depression of the fields may also be due to impaired cerebration depending on physical enfeeblement or disease, such as cerebral arterio-sclerosis. Field changes of this type may be encountered when the examination makes relatively heavy demands upon the faculties of the patient, as, for example, when certain elderly patients are tested with small visual angles. The possibility of this causation should be borne in mind, and such changes should not be confused with the effects of psychical disorder on the one hand or those of disease of the visual path on the other.

Alterations in the visual fields are of great value in the diagnosis of functional conditions and are regarded as pathognomonic by some observers. Perimetry is also useful in prognosis as the state of the fields tends to vary with the condition of the patient. It should, however, always be remembered that there is no essential difference between neurasthenia and hysteria and that the field changes are in reality determined by suggestion in both conditions. Thus there is no special field change which is typical of functional conditions, as Roenne points out, what appears to be typical is merely the consistent way in which the hysteric reacts to the same form of examination. This conception also explains the varying results which may be obtained by different observers—each tends to get his own reaction.

Neurasthenia

In neurasthenia or anxiety neurosis or traumatic neurosis the fields are characterised by instability and exhaustion. The field usually appears somewhat contracted to begin with, and during the examination soon shrinks further, and if the test be recommenced with a much larger object the field may be a little wider at first, but rapidly contracts again. Thus, if the test-object is moved radially and centripetally in successive

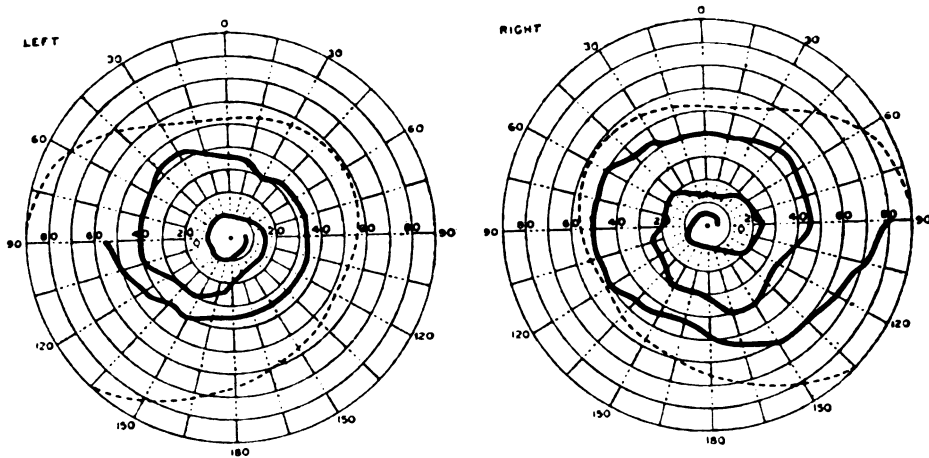


FIG. 245.—SPIRAL FIELDS. WAB NEURASTHENIA. CONTRACTING SPIRAL. (HEALY.)

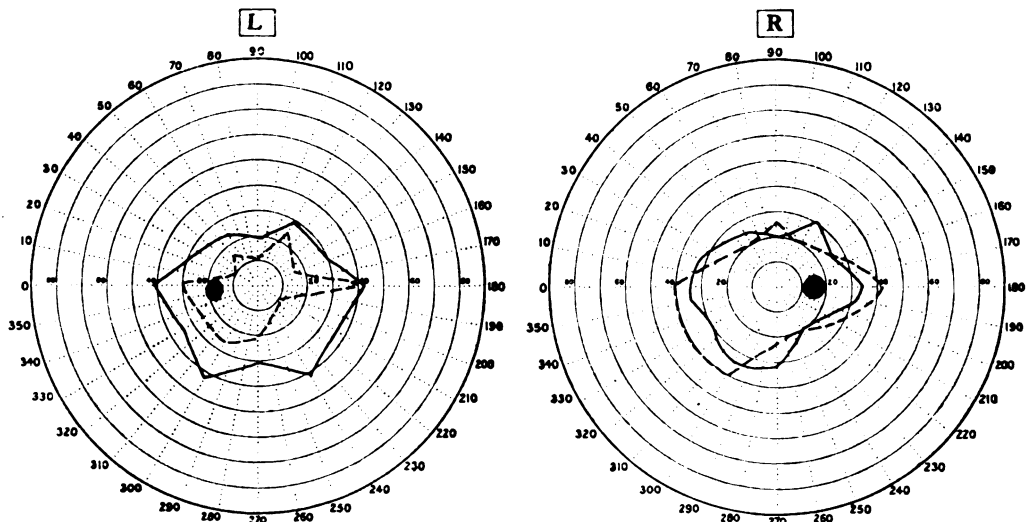


FIG. 246.—FUNCTIONAL FIELD CHANGES. STAR-LIKE AND INTERLACING FIELDS WITH PRONOUNCED CONTRACTION IN A NEUROTIC FEMALE PATIENT.

Objects $2\frac{1}{2}$ 0, $2\frac{1}{2}$ 6. R.V., L.V. 5. (A., 1915.)

meridians, continuing the process until a constant result is obtained for each meridian, a contracting or inward spiral is produced on the chart terminating in a more or less small central field. If the two opposite ends of the vertical and horizontal meridians be tested and then the ends of the intermediate meridians (45° and 135°), and the eight resulting points joined, the figure on the chart resembles a four-pointed star. Or the ends of a single meridian, the horizontal for preference, may be successively tested when it will be found that the limits at each side become more restricted at each trial.

It is characteristic that when one field has been tested to the limit of reduction the field of the other eye will be found to be already contracted, and this may be verified on a subsequent occasion by testing the fields in the reverse order.

The classical "exhaustion" or "fatigue" fields and "oscillating" fields belong to the same group of phenomena, and have in themselves no special value or significance apart from the indication of functional disturbance which they afford. The oscillating variety, in which the test object appears and disappears at intervals along the various meridians, may be elicited in a more elaborated form as multiple concentric ring scotomata. This might almost be compared to a stammering of perception and occurs always at the periphery of the field (whether contracted or not), never in the central area.

The extent of the field may be enlarged by stimulating the patient to try hard and by other suggestive measures, so that an expanding instead of a contracting spiral may be produced. These contrary spirals were noted by Fuchs in 1900, and have been noted by other observers more recently. Hurst and Symms point out that they can be produced by moving the test object centrifugally. Von Reuss found the spirals more easily elicited with white than with colour and found blue the best colour for the purpose.

In neurasthenia the patient is partially suggested, and the field changes are mobile and alter during examination. The evidence of expanding spirals, and of the result of testing one field upon the field of the other eye, shows that there is no retinal fatigue and that the symptoms have a central origin.

Many neurasthenics, especially those with traumatic neurosis, are very suggestible and subconsciously, if not also to some extent consciously, anxious to display some kind of defect or peculiarity in response to every form of examination.

Fields of this type, though less pronounced in degree, are not infrequently found in patients who are in any way enfeebled or "run down" without being definitely neurasthenic in the sense usually understood.

Miners' nystagmus is a condition which, even though possibly not of purely psychogenic origin, presents a symptom complex of definitely psychogenic type. The fields of vision show all the characters described above and the examination of the fields was advocated by Cridland to estimate the completeness of recovery after the objective symptoms had disappeared. In cases of neurasthenia among soldiers which present similar symptoms but without the nystagmus the estimation of the severity of the affection by testing the rapidity and amount of the field contraction was proposed by Healy. In both cases, however, the results obtained will depend to a considerable extent on the personality of the examiner.

Hysteria

The typical field change found by ordinary perimetric examination is concentric contraction, which remains more or less unaltered in extent when examined at different distances or with objects of different sizes. The field is, therefore, tubular in type, a form which is necessarily of subjective origin. The defect is present when the examination begins, and is usually constant while the test is proceeding, unless altered by suggestion, but tends to vary considerably on different occasions. Although the fields may be extremely contracted, central vision is often good, or, in other cases, any degree of amblyopia may be present. When no determining cause can be discovered, as is

often the case in female subjects, the contraction is usually bilateral and symmetrical. When one eye has been injured the extent and intensity of the defect may be different in the two fields. A spurious hemianopia may be present, the patient being convinced that he must be blind towards the side of the real or supposed defect. The fact that compensation is paid for the loss of an eye often impresses a subject (*e.g.*, workman) that there must be a defect towards that side, as he argues that he is not being paid compensation for nothing. The binocular field may be affected, although one uniocular field is nearly or quite intact (*e.g.*, homonymous hemianopia binocularly with intact field of one eye when tested alone).

The extent of the contraction varies, but is usually considerable and often extreme,

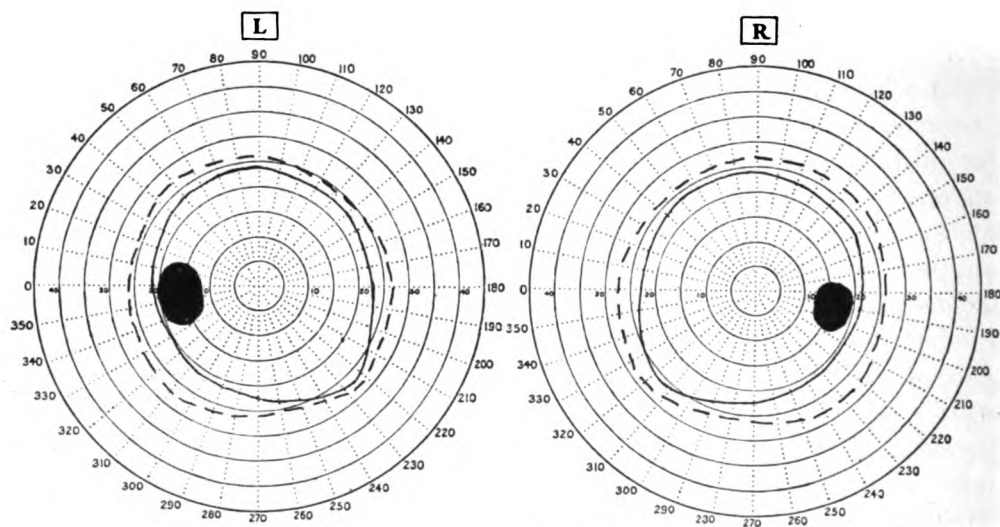


FIG. 247.—ENLARGEMENT OF BLIND SPOT IN HYSTERIA.

R.V., L.V. $\frac{5}{2}$. Test-objects $\frac{5}{2} \frac{0}{0} \frac{0}{0}$ - - -, $\frac{5}{2} \frac{2}{0} \frac{0}{0}$ —. The field for $\frac{5}{2} \frac{0}{0} \frac{0}{0}$ is approximately the size of the normal $\frac{5}{2} \frac{1}{0} \frac{0}{0}$ field and that for $\frac{5}{2} \frac{2}{0} \frac{0}{0}$ only slightly larger. The blind spots are greatly enlarged. (W., 1932.)

so that the field covers only some 10° to 30° . The blind spot may be enlarged, its margin behaving in the same way as the peripheral limit of the field. The edge of the field is very steep, and enlarging the test-object does not increase its extent more than a little if at all. The patient may be very definite about this feature, and may say that only half of a large object, such as the hand or a sheet of writing paper, is seen when it is held at the alleged boundary. This steepness of the edge has sometimes been mistakenly interpreted as indicative in itself of an organic lesion. It is, on the contrary, extremely suggestive of a functional cause in cases in which, on other grounds, functional disturbance is suspected.

The fields in hysteria present evidence of more complete suggestion than in neurasthenia, and, as Roenne, Hurst and Symns and others have pointed out, there is no characteristic perimetric sign, the changes resulting from the patient's imagination together with the method of examination.

The following cases are typical examples :—

I.—Mrs. C. G., age 25, married three years, no children. Four weeks previously headaches and bad vision which have continued. Examination disclosed no objective signs of disease of any kind. Pronounced tenderness on pressure was found over both eyeballs; vision varied from $\frac{6}{9}$ to $\frac{6}{18}$ on different occasions. Both fields were greatly contracted: At 330 mm. with 30 mm. object field about 30° ; with 60 mm. about 35° , this object coming bit by bit into sight as it crossed the field limit; an effort at suggestion then increased the 30 mm. field to about 45° , and subsequently the 60 mm. field was found to be even smaller than before. On the screen at 2,000 mm. a sheet of paper, about 10 inches square, was not seen until within 20° of the fixation point. No loss of orientation. In this case no cause could be discovered.

II.—Mr. W. L. complains of a mist before his right eye since an injury to the cheek just below the eye three years previously. Both eyes quite normal objectively. Vision: left eye, $\frac{6}{8}$, field normal; right eye, hand movements close to face, field much restricted for large objects. The binocular field showed right hemianopia. Suggestion was successful in producing complete cure in about ten minutes.

Diagnosis.—It is essential that the perimetrist should be familiar with functional field defects and able to recognise them. They are easily produced both in normal and pathological conditions, and are often mistaken for genuine evidence of the presence of organic lesions. The best method for the student of perimetry is to devote a little time with a susceptible subject to practising the production of such field changes by the use of suggestion. He will soon appreciate the ease with which he may slip into or avoid pitfalls of this kind. In regard to a subject of this nature, it is impossible to lay down hard and fast rules, and the observer must rely largely on his experience and clinical insight.

The most important factor in the diagnosis of these field changes is the recognition of inconsistencies in the defects themselves, and between them and the other features of the case. The history, mode of onset and course of the defects are often helpful. Before testing the fields central vision should be examined with different sizes of type and at different distances to elicit discrepancies and inconsistencies. Pronounced contraction of both fields with normal optic discs and no loss of orientation is very suspicious. Severe depression or blindness of one field, the other being intact when tested alone, associated with depression extending into the paired portion of the binocular field, in the absence of an evident lesion or abnormality, is conclusive evidence of the presence of functional disturbance though not exclusive of an organic defect in the blind eye. The binocular field should always be tested.

The use of the quantitative method can practically always be relied upon to clear up even the most doubtful case. The fields should be examined with perimeter and screen and different sizes of test-objects. Roenne (336) recommends a 10-mm. object at 300 mm. followed by a 68-mm. object at 2,000 mm. The visual angle is the same in each case, and in organic disease the corresponding fields cover approximately the same extent in degrees. Or a much smaller visual angle may be used with the perimeter, $\frac{2}{300}$, for example. Nevertheless the screen test nearly always gives a smaller field in functional cases, though sometimes, when the perimeter field is very small,

the screen field—with a large object, but the same or even a smaller visual angle—may be larger. Tubular fields are well demonstrated with the screen. Repetition of the test after a short interval to determine the constancy of the findings is of value, especially when temporal contraction, interlacing of the colour fields, multiple concentric ring scotomata and the like have been elicited. On the second occasion the fields should be tested in the reverse order, so as to note the effect of testing one field upon the other. Contraction induced in this way is a valuable diagnostic sign.

Steepness of the edge of the field in concentric contraction, the test-object appearing suddenly or, if large, bit by bit as it crosses the boundary, should arouse suspicion that the defect is functional.

If it is attempted in a case of functional contraction to map out several isopters for white and colour so as to show the slope of the field, difficulty and inconsistency are always encountered, whereas the process is usually easy in organic disease.

When the patient professes almost complete blindness the remnant of vision is always central, never peripheral, and the condition superficially resembles the severe stage of quinine poisoning. The history and the other signs of cinchonism make differentiation easy.

Great care must be taken to avoid involuntary suggestion, while at the same time the skilful use of suggestion to alter the field may be most valuable. More difficulty is presented by cases in which a functional element is associated with or has supervened upon an organic lesion such as injury or multiple sclerosis, and genuine field changes may be exaggerated or distorted or new ones invented, but careful examination on the lines indicated will enable the observer to avoid misdiagnosis. Neglect to examine the fields thoroughly by the quantitative method leads to inadequate demonstration of the characters of the defects, and, therefore, to misinterpretation. The organic origin attributed to the so-called "bitemporal contraction" of pregnancy and to the concentric contraction described in chronic nasal sinus disease, and the "enlargement" of the field following the administration of strychnine, still mentioned in modern text-books of pharmacology, may be regarded as instances of the results of this omission. Although central scotoma is never simulated by neurotics it can be acquired by squinting children. When the young hypermetrope with inadequate fusion sense is faced with the alternative of diplopia or suppression of one image he finds himself able to adopt the latter as a means of escape and, since only central and paracentral vision cause annoyance, only a central scotoma is acquired. The scotoma is central and approximately circular in shape or oval, extending towards the blind spot with its long axis horizontal, and is always relative. Its size and intensity vary with the degree of depression of vision. When vision is $\frac{6}{60}$ or better the scotoma is small, extending 2° or 3° from the fixation point, if the vision is worse the scotoma is larger and more dense. In extreme cases the peripheral field may also be affected. The gradual development of the defect and its curability—at least in its early stages—by covering the other eye are characteristic features. This functional scotoma is the basis of the so-called *Amblyopia ex Anopsia* and is of interest to the perimetrist in so far as its demonstration may be of use in the diagnosis of obscure cases of amblyopia,

especially those with little or no refractive error and no history of squint. (See Peter, 312; Uhthoff, 432; Worth, 478.)

In suspected malingering examination of the fields with different visual angles and at varying distances will often be found useful. The position of the blind spot may be utilised (von Szily) for the detection of simulated unilateral blindness. If a test-object be placed on the blind spot of the seeing eye, both eyes being open, it can only be seen there by the supposedly blind eye. The test should be conducted at such a distance, say 2 m., that the projection of the blind spot is large, and it is obvious that the co-operation of the patient, not to be relied upon in such cases, is essential in regard to steady fixation. The exposure of malingering is, however, no part of the legitimate business of perimetry, which in this case becomes merely another dodge of the examiner to entrap the ignorant and unwary.

APPENDIX

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I. Isopters in the Normal Field

A. THE FIELD FOR WHITE

Observer.	Angle in minutes.	Object in millimetres.	Distance in millimetres.	Field in degrees.			
				Out.	Down.	In.	Up.
Traquair	0.86	1.0	4,000	8.3	7.4	8.5	5.3
Roenne	1.0	1.25	4,000	10.0	8.0	6.0	4.0
Walker	1.72	1.2	2,500	20	18	17	17
Sinclair	1.72	1.0	2,000	26	25	26	24
Hefftner	1.72	1.0	2,000	22	18	18	15
Walker	2.1	1.2	2,000	30	26	25	24
Roenne	2.1	2.5	4,000	10	13	15	11
Walker	4.3	1.2	1,000	51	43	40	47
Roenne	4.3	2.5	2,000	33	27	31	24
Meisling	5.15	3.0	2,000	30	30	30	25
Hefftner	5.15	3.0	2,000	32	29	25	26
Sinclair	5.15	3.0	2,000	37	30	30	25
Igersheimer	6.9	2.0	1,000	48	36	39	36
Roenne	8.6	5.0	2,000	72	51	50	51
Feree, Rand and Monroe	10.2			64	46	47	38
Meisling	10.3	6.0	2,000	50	40	40	35
Sinclair	10.3	6.0	2,000	50	40	40	35
Traquair	10.4	1.0	330	80	59	57	48
Waldeck	11.46	1.0	300	70	55	55	51
Walker	15.0	1.2	286	90	64	60	52
Roenne	17.2	5.0	1,000	82	69	60	66
Traquair	20.8	2.0	330	87	65	60	50
Waldeck	23.0	2.0	300	83	60	58	58
Roenne	34.3	10.0	1,000	93	76	62	69
Hefftner	34.4	3.0	300	70	55	56	50
Feree, Rand and Monroe	60			90	70	60	58
Hefftner	229	20.0	300	96	66	59	54
Roenne	275	80.0	1,000	104	76	62	69

THE isopters found for the same or almost the same visual angle by different observers vary considerably. By making use of variations in the relationship between the size of the test-object and its distance from the eye the same angle may be obtained in several different ways, and each writer has used his own method. The Table on p. 288 shows the angle, the object and distance used, and the resulting field. The figures are arranged in groups corresponding to visual angles of somewhat similar size; those given for Waldeck, who used square objects, are averaged from his findings.

An examination of these data shows that, as a rule, where different observers have used similar visual angles, the diameter of the test-object and its distance from the eye being approximately the same in each case, the results obtained agree fairly closely. On the other hand, where the same visual angle has been obtained by larger or smaller objects associated in each case with the appropriate longer or shorter distance the field is larger for the shorter distance. For example, $\frac{2}{3} \frac{1}{10} \frac{1}{10}$ gives practically the same angle as $\frac{3}{4} \frac{1}{10}$, yet the field for the latter is considerably larger; the field for $\frac{2}{3} \frac{1}{10} \frac{1}{10}$ is larger than that for $\frac{4}{5} \frac{1}{10}$, and so on. This relationship, however, only holds good for the smaller visual angles, and is more noticeable the more the angle becomes reduced, i.e., the more difficult the object is to see. On this ground it may be regarded as due to the reduction of illumination which occurs when the distance is increased and which is evidently insufficient to produce much effect except in the case of small objects. No doubt it is this quality of the screen examination at 2,000 mm. which helps to make it an efficient test in, for example, retinal detachment.

B. THE FIELD FOR COLOURS

The isopters for colours given in the Table on p. 12 represent averages from six subjects, three male and three female, examined in ordinary winter daylight with colours of the type commonly obtainable in paper.

For the most part, the mean in each case lay nearer the smaller than the larger of the extremes, so that the figures given represent a high rather than a low estimate. The difference between the extremes was often great, especially when the test was difficult, as with small visual angles in red and green. With the larger angles the findings often lay very close together. This is a reflex of the difficulty experienced by the subject in giving what is considered to be an accurate response, and every perimetrist should have his own field tested with small visual angles in colour, in order to appreciate the patient's position.

A few control tests carried out with blinds drawn or in the afternoon showed that except in the case of very small visual angles individual differences were much greater than any that could be attributed to variations in illumination such as are commonly met with. A not too bright light proved more satisfactory and made the examination easier since, with strong light, red and green objects moved centripetally, took longer to appear coloured. This again affected the smaller angles more especially, and the colour, green, whose white value was too high. The relationship found between the distance of the test-object and the size of the field in the case of white objects was present only to a slight degree for colours except on the temporal side of the field, where it was pronounced, a much larger field being obtained at the shorter distance, the visual angles used being approximately equal in each case. For example, $\frac{3}{4} \frac{1}{10}$ (angle 20.8') gave a field for each colour nearly equal to, in some meridians smaller than, that for $\frac{1}{2} \frac{1}{10} \frac{1}{10}$ (angle 17.2') except on the temporal side, and here the difference was pronounced for blue, slightly less for red and greatly reduced for green. This suggests that, in this case, the increase in illumination produced by reducing the distance is responsible for the small fields at the short radius by rendering the hue less distinct through accentuation of the white element. It was also noticed that a dark glass increased the field for green for the small visual angles at 330 mm. More research is required in connection with the isopters both for white and colours, but the conclusion seems clear that with the ordinary tests, both white and coloured, and in the usual circumstances of the consulting-room or hospital clinic, we are not entitled to assume that equal visual angles give more than approximately equal fields.

All the subjects found that as the object travelled from periphery to centre it became coloured, lost its colour, then regained it, the hue increasing in intensity up to the fixation point—that is to say, for each colour and for each size of object there was a ring scotoma at the periphery of the field for that visual angle. The figures are, of course, taken from the point of permanent appearance of the colour. It is possible that an exaggerated degree of this functional ring scotoma may be the underlying factor in connection with some of the reported forms of peripheral ring scotoma.

For comparison, the data found by some other observers may be quoted here. The following figures are from various sources :—

C.F.

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Observer.	Angle.	Object (mm.)	Dis- tance (mm.)	Blue.				Red.				Green.			
				Out.	Down.	In.	Up.	Out.	Down.	In.	Up.	Out.	Down.	In.	Up.
Peter.	52°?	5	330 ?	65	48	39	39	40	30	25	26	30	25	19	18
Hirschberg.	...	10	...	62	54	42	40	52	47	32	28	32	23	20	20
de Schweinitz	2° ?	10 ²	300	80	58	45	40	65	45	30	33	50	30	25	27
Landolt	...	10 ²	...	90	70	56	56	84	56	37	38	78	49	27	32
Baas	...	20 ²	...	84	62	50	48	75	50	40	39	63	43	33	32

Roenne found the field for $\frac{10}{300}$ red (114') equal to or rather less than that for $\frac{10}{2000}$ white (17'), that is, nearly the maximum field.

In the binocular field it was found that the figures were slightly larger for the paired portion but smaller, if anything, for the unpaired part for small white objects as well as for colour. It was found more difficult to detect colour at the temporal periphery if the eye not concerned was open than if it was shut. No evidence was found in support of "binocular intensification" in the sense indicated by Gowers, *i.e.*, temporal enlargement of the field owing to the unstimulated eye being open. As regards the sexes the fields appeared to be very nearly equal. The figures for the females were rather higher at 2,000 mm., and those for the males at 330 mm., and no indication was found that one sex had larger colour fields than the other.

Although examination with small visual angles in white can provide much of the information to be gained from colour testing, the latter is a useful clinical method and often of great service. Its value and reliability in the investigation of disease is much greater than can be estimated from a study of the results obtained under normal conditions.

For information regarding "physiologically pure" colours and the clinical results obtained by their use, the papers by Engelking, Engelking and Eckstein, and Aulamo (14, 97-100) may be consulted.

II. The Blind Spot

1. POSITION, SHAPE AND DIMENSIONS

Observer.	Distance from Fixation Point.	Distance of Centre below Horizontal.	Vertical Diameter.	Horizontal Diameter.
Van der Hoeve	15° 33' 47"	1° 40' 41"	7° 26'	5° 42' 55"
Peter	15° 49'	1° 30' }	7° 40'	5° 28'
Traquair	15° 27' 36"	1° 24' } (Approx.)	7° 17'	5° 7' 5"

The position of the blind spot, somewhat below the horizontal meridian of the visual field, corresponds to a position of the optic disc slightly above—not below as sometimes stated—the horizontal meridian of the eye. It is legitimate to infer that the position of the optic disc, when the eye is in use, is above the horizontal meridian. This is its physiological position and it may be presumed that its anatomical position is nearly the same.

The oval outline does not correspond to the shape of the optic nerve head as seen in the dissected eye. No doubt the vertical elongation is referable to the presence of the large vessels at the superior and inferior poles of the disc though it is not certain that this is the sole cause.

2. THE AMBLYOPIC ZONE

This narrow marginal band of relative blindness surrounding the blind spot was originally demonstrated by Bjerrum. Its width and intensity are not yet definitely established. Sinclair, using a 1 mm. white object at 2 m. (V. Angle = 1.7') found this zone to be approximately 1° wide, and also demonstrable with colours. Van der Hoeve, using larger visual angles, found a zone of $\frac{1}{8}^{\circ}$ to $\frac{1}{4}^{\circ}$ of relative blindness for

white, and $\frac{1}{2}^{\circ}$ to $\frac{3}{4}^{\circ}$ of relative blindness for colours. Haycraft, who used colours of equal luminosity, found a much wider area of modified colour perception. In this amblyopic zone he found that colours when moved centrifugally from the blind spot were recognised in the order blue, yellow and green, and lastly red, the same colours being perceived in the same order when moved centripetally at the periphery of the field. Thus the outer periphery of the field corresponds in this respect to the margin of the blind spot. Haycraft does not state the visual angles used.

The writer's observations agree with those of Sinclair. Using the screen at 2 or 4 m., the blind spot being first marked out with a 10 mm. or 20 mm. object, the amblyopic zone is easily demonstrated with a 1 mm. or 2 mm. white test respectively. With $\frac{20}{1000}$ it is somewhat narrower than with $\frac{20}{100}$. With these visual angles it is rarely wider than 1° except at the upper and lower margins of the blind spot. The discrepancies found by various observers are attributable to the different methods of examination used. The amblyopic zone is independent of the physiological "travel of fixation." This can be demonstrated by comparing the margin of a steep-edged absolute scotoma with that of the blind spot in any case in which such a scotoma, a normal blind spot, and good fixation are present.

Measurements have also been made by Berens, Marlow, Ineze, Eichenberger, and Feree, Rand and Wentworth whose papers may be consulted: an exhaustive review of the subject is contributed by Brøns (43).

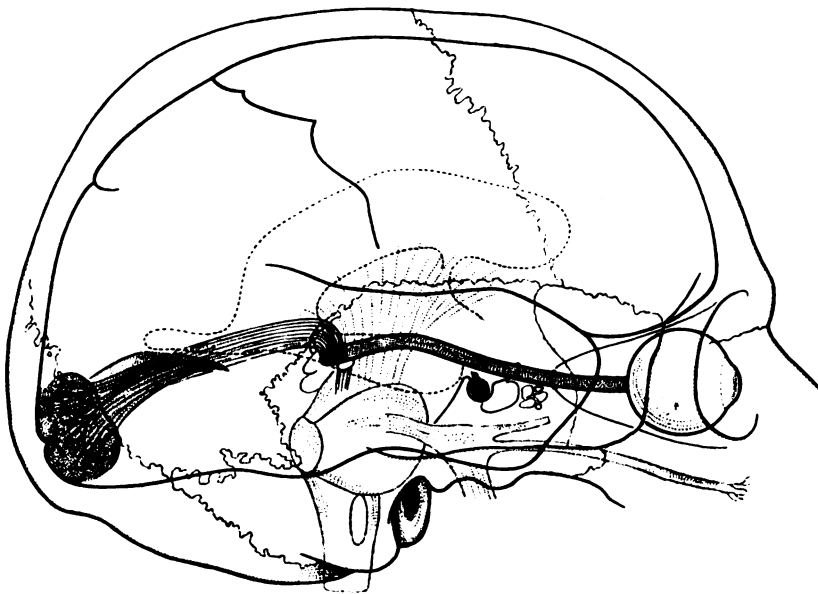


FIG. 248.—SCHEME OF THE VISUAL PATH SHOWING THE OPTIC RADIATION ACCORDING TO HENSCHEN.
(With acknowledgements to the *Edinburgh Medical Journal*.)

III. Anatomical Relations of the Visual Path

A brief survey of some anatomical features which influence the causation of field changes may prove convenient to the reader.

The optic nerve leaves the eyeball at a point about 15.5° to the medial side of the macula lutea and slightly above the horizontal meridian. Its course to the optic foramen is slightly sinuous so that it may undergo considerable displacement and deformation without functional interference.

As it enters the optic foramen the optic nerve is usually related to a posterior ethmoidal cell on its medial, or upper and medial side. In the canal the relationship is, as a rule, to the sphenoidal sinus of its own side, but may be varied so that either or both sphenoidal sinuses may be concerned. An ethmo-sphenoidal or anterior sphenoidal cell may almost surround the optic canal. The canal may be formed of thick or thin bone, or merely membrane at some part, and its contact with the air cells may be very slight, or it may be related on all sides to one or more.

Within the canal the membranes covering the nerve and the periosteum are closely bound together, and as the nerve emerges into the cranial cavity it passes abruptly from a state of fixation to one of mobility

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and is covered above by a horizontal sharp-edged fold of dura mater continuous with the edge of the tentorium.

Above the intracranial part of the nerve is the frontal lobe, and below and laterally lies the carotid trunk which is here acutely folded upon itself in an antero-posterior direction so that the upper part, passing

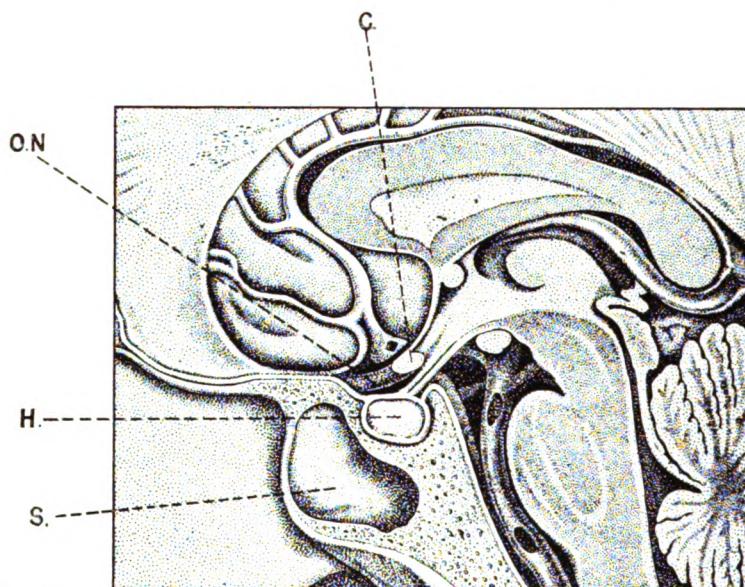


FIG. 249.—ANATOMICAL RELATIONS OF THE OPTIC CHIASMA. C. CHIASMA. O.N. OPTIC NERVE. H. HYPOPHYSIS. S. SPHENOIDAL SINUS.

backwards under the nerve, lies almost parallel to the lower part which passes forwards in the cavernous sinus lateral to the hypophysis. From the upper part of this bend, after it has penetrated the dura and arachnoid, the ophthalmic artery passes into the optic canal beneath the nerve and is closely applied to it within its arachnoid and dural sheaths which it leaves as it enters the orbit, and then crosses over the nerve from the lateral to the medial side. While in contact with the artery the nerve may be deeply indented by it and even split into two parts. There is no evidence that this anatomical anomaly has any effect upon the visual field.

The carotid artery having passed backwards from the optic foramen below the nerve turns upwards at the lateral side of the junction of the nerve and chiasma and divides into the anterior and middle cerebral branches. The former turns sharply above the chiasma or nerve towards the median plane and is connected with the opposite anterior cerebral by the anterior communicating artery which lies above the interval between the optic nerves. The posterior communicating branch passes on each side from the carotid trunk beneath the optic tract to the posterior cerebral artery so that the chiasma and its limbs are embraced by the *circulus arteriosus* (Circle of Willis). Numerous variations and anomalies of the arterial circle occur so that the results of morbid conditions of the vessels need not be always the same, as the position of the branches and the efficiency of the collateral circulation may vary in different cases.

From the posterior ends of the optic canals the optic nerves, becoming here transversely oval or rather pear-shaped in section with the rounded side medially, slope backwards, medially, and upwards to the chiasma, where they meet at a comparatively acute angle. The intracranial portion of the nerve varies greatly in length from about 4 mm. to 21 mm., averaging 13 mm., and the angle of junction with its fellow is wider when the nerve is short. The chiasma lies above and behind the *tuberculum sellæ* of the sphenoid, only rarely does its anterior border lie close to or in the optic groove. The body of the chiasma lies over the posterior half or two-thirds of the hypophysial fossa and is separated from the *diaphragma sellæ* by a vertical space of 5 to 10 mm., rarely less. Its posterior border extends usually (79 per cent.) slightly behind the level of the *dorsum sellæ*, occasionally (4 per cent.) its whole body lies behind the fossa and over and behind the *dorsum sellæ* (post-fixed chiasma). In 5 per cent., the chiasmal body lies anteriorly on the optic groove (prefixed chiasma), and in 12 per cent., it is situated directly over the hypophysial fossa (de Schweinitz, 85). There is, therefore, a free triangular space in front of the chiasma, bounded in front by the

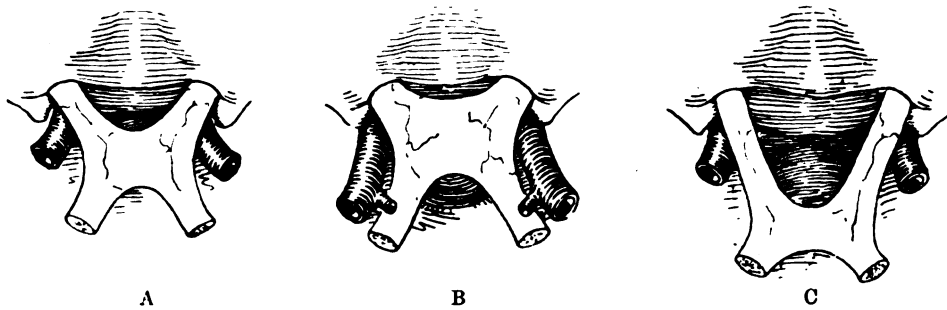


FIG. 250.—THE RELATION OF THE CHIASMA TO THE SELLA TURCICA.

A. The common position, the chiasma lying over the posterior part of the sella, 79 per cent. B. Prefixed chiasma, 5 per cent. C. Post-fixed chiasma, 4 per cent. (After Schaeffer.)

sphenoid and laterally by the optic nerves, in which the anterior half or even more of the hypophysis is exposed covered by the diaphragma. Rising from the foramen in the posterior part of the diaphragma, the hypophysial stalk arches upwards and backwards over the dorsum sellæ, passing into the infundibulum of the third ventricle. The infundibulum is in close contact with the lower posterior part of the chiasma which it joins at a very acute angle. The chiasma lies in the anterior part of the cisterna basalis of the subarachnoid space and forms the floor of the optic recess of the third ventricle which extends almost to its anterior border, so that the chiasma is suspended in and surrounded by the cerebro-spinal fluid except at its posterior margin. The relationship of the neighbouring arteries has been mentioned. Close above and to the lateral side of the anterior angle of the chiasma lies the olfactory lobe and below its side is the cavernous sinus with its contained nerves, of which the third lies below the lateral edge of the chiasma before it enters the sinus. Between the optic nerves the arachnoid is spread across like an apron, and it is attached to the tip of the temporal lobe and to the carotid artery at each side and to the frontal lobes anteriorly. It closely surrounds the infundibulum and in the angle between this structure and the chiasma a dense vascular network of pia-arachnoid is found. The chiasma is thus surrounded by a membrane which holds it up to the brain and which becomes densely matted and thickened in inflammatory conditions, notably syphilis.

As the tract leaves the chiasma it is free except on the medial side where it is attached to the lateral wall of the optic recess of the third ventricle by a narrow band. Passing backwards, laterally, and slightly upwards it appears a little twisted as it winds round the cerebral peduncle, against which it is flattened and to which it is closely adherent although both edges now appear free. The superior fasciculi are partly surrounded by the commissures of Meynert and Gudden while the inferior bundles are free and covered by thin pia mater. More posteriorly the tract passes below the internal capsule and the globus pallidus of the lentiform nucleus and it is situated in the superior part or roof of the transverse fissure lateral to the basis pedunculi, covered on its lateral and inferior aspect by the amygdaloid nucleus, the uncus and the hippocampal gyrus of the temporal lobe. These relationships are of interest in connection with tumours in this area.

Beneath the thalamus the tract gives off a smaller medial portion, while the larger lateral portion, which contains the visual fibres, passes to the lateral geniculate body, where most of the fibres end; a few, which are not visual fibres, pass on in the superior brachium to the superior corpus quadrigeminum.

The lateral geniculate body lies on the side of the cerebral peduncle on the most lateral part of the lower surface of the pulvinar covered infero-laterally by the hippocampal gyrus.

From the lateral geniculate body the fibres of the optic radiation pass outwards into the retrolenticular part of the posterior limb of the internal capsule, where they lie behind the sensory fibres and medial to the auditory bundle. Running downwards parallel to and curving round the lateral wall of the posterior horn of the lateral ventricle, as a compact bundle 5 to 10 mm. in height, they then turn backwards lying deeply in the white matter of the posterior part of the temporal lobe about the level of the middle temporal gyrus. Farther back this bundle lies along the infero-lateral angle of the posterior horn of the ventricle beneath which it extends to the occipital cortex. In the occipital lobe the visual fibres form a vertical sheaf, anteriorly about 20 mm. in height and expanding posteriorly, the dorsal fibres passing to the upper lip of the post-calcarine sulcus and the ventral fibres to the lower lip of the post-calcarine and calcarine sulci. The floor of the sulcus receives both superior and inferior fibres and in the occipital pole the fibres spread out fan-like to the cortex.

Henschen points out that the term "optic radiation" is often used loosely to include fibres other than the centripetal visual fibres.

The study of horizontal hemianopia following occipital wounds involving both sides has suggested (Holmes, Roenne) that the fibres related to the upper and lower retinal quadrants are separated in the radiations by an anatomical interval which contains the macular bundle. Cases recorded by other observers also lend support to this view.

A description of the optic radiation, founded on pathological anatomical observations, differing in important details from that of Henschen, is given by Adolf Meyer (286, 287), and illustrated by diagrams which appear in papers by Cushing (69) and Pfeiffer (314). Meyer divided the radiation into dorsal, lateral and ventral bundles. The dorso-lateral bundles pass

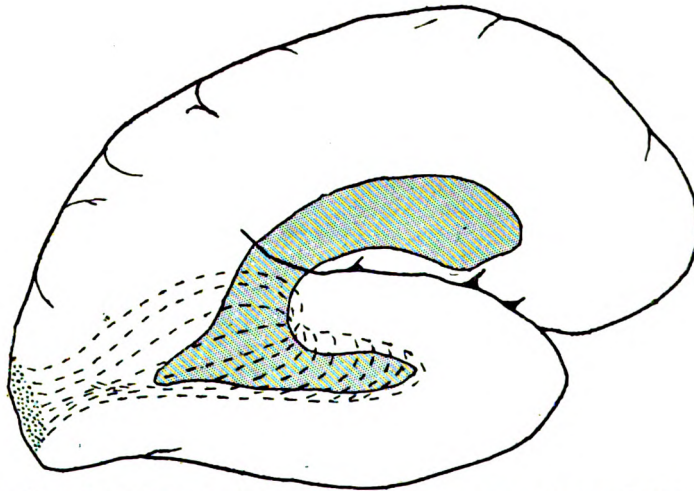


FIG. 251.—THE OPTIC RADIATION ACCORDING TO MEYER, SHOWING THE TEMPORAL LOOP.

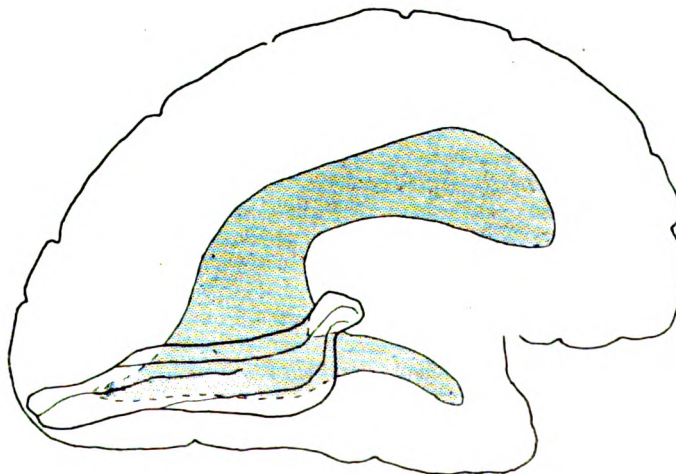


FIG. 252.—THE OPTIC RADIATION ACCORDING TO ADROGUÉ.

directly to the occipital visual cortex, the ventral fibres are directed downwards and forwards into the uncinate region of the temporal lobe, forming a *détour* or loop which passes over and round the inferior horn of the lateral ventricle, then turning sharply backwards along the inferior aspect of the ventricle to the anterior part of the inferior calcarine cortex. The diagrams represent the most ventral fibres of this loop as extending forwards nearly to the tip of the ventricular horn, to within a short distance of the level of the posterior margin of the chiasma.

This description differs materially from that of Henschen in that it represents the visual fibres as fanning out widely immediately they leave the lateral geniculate body to such an extent that they cover the outer and inferior aspect of practically the whole of the lateral ventricle below the point where it turns downwards into the temporal and occipital lobes, whereas according to Henschen—and the hitherto generally accepted view—they form a relatively small compact bundle in the anterior part of their course.

These inferior fibres correspond to the inferior retinal quadrants, and, therefore, lesions of the anterior part of the temporal lobe, if sufficiently deep, imply a homonymous superior quadrant hemianopia of the opposite side, the presumption being that the site of interference is in the temporal lobe, i.e., suprageniculate. Cushing clearly and beautifully demonstrated the nature and quality of the homonymous hemianopia in temporal lobe lesions, but the clinical evidence brought forward does not exclude a subgeniculate, i.e., tract, site of interference; in fact, in several respects, it favours this explanation (Traquair, 401).

The existence of visual fibres in Meyer's loop is not accepted by the Buenos Aires school: "El concepto de la cinta o lamina óptica de Pfeiffer es también erróneo. No existen fibras ópticas ni en la rodilla temporal ni tampoco en el Zípfelmütze." (Adrogué, 5, p. 11.) (Fig. 252.)

The cortical visual area occupies the lower lip of the calcarine sulcus, the floor and lips of the post-calcarine sulcus and the tip of the occipital pole extending probably only to a slight extent on to the visible lateral surface of the occipital lobe.

Recent research has established with probable accuracy the projection of the retina upon the occipital cortex.

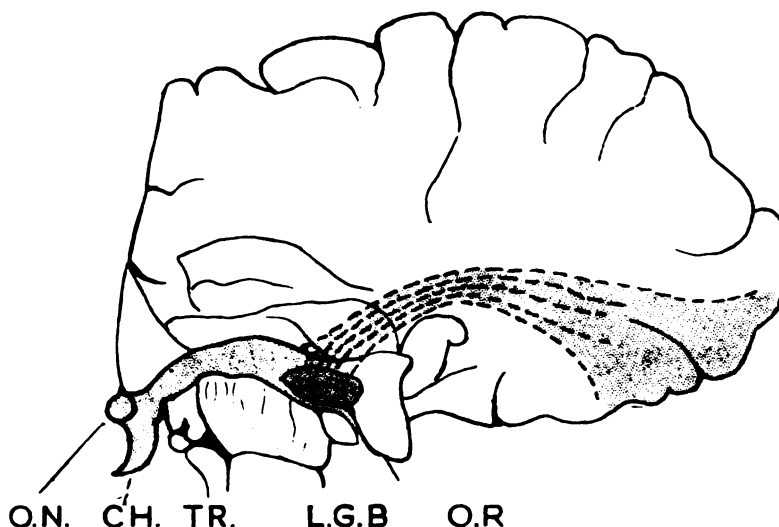


FIG 253.—THE VISUAL PATHWAY ACCORDING TO MALBRAN (277).

O.N. Optic Nerve. Ch. Chiasma. Tr. Tract. L.G.B. Lateral Geniculate Body. O.R. Optic Radiation.

The following are some of Holmes' conclusions, which he, however, does not regard as final :—

- “(1) The upper half of each retina is represented in the dorsal [superior], and the lower in the ventral [inferior] part of each visual area.
- “(2) The centre for macular or central vision lies in the most posterior part of the visual cortex, probably on the margins and on the lateral surfaces of the occipital poles. The macula has not a bilateral representation.
- “(3) The centre for vision subserved by the periphery of the retina is situated in the anterior portions of the visual areas, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forwards in the visual cortex.
- “(4) Those portions of the retina adjoining their vertical axes are probably represented in the dorsal [superior] and ventral [inferior] margins of the visual areas, while the retina in the neighbourhood of its horizontal axis is projected on to the walls and the floor of the calcarine fissure.”

Wilbrand and Saenger, who believe the macula to have a bilateral cortical representation, place the macular centre 'no quite as far back as the tip of the occipital pole, the most posterior part of the visual area containing the centre upon which depends the macular sparing when the opposite visual centre is completely destroyed. The most anterior part represents the unpaired area at the temporal periphery of the field of the opposite eye. The radiation is arranged so that the fibres for the unpaired area, which form a separate bundle, lie medially and those for the area of macular sparing laterally. Lenz believes that the two macular centres are connected by commissural fibres. Penfield, Evans and MacMillan (308) also found that excision of the posterior part of the temporal lobe causes homonymous hemianopia with division of the central area whereas in hemianopia after removal of the occipital lobe the central area is spared. If, however, the anterior limit of the incision extends as far forwards as the splenium the central area is bisected.

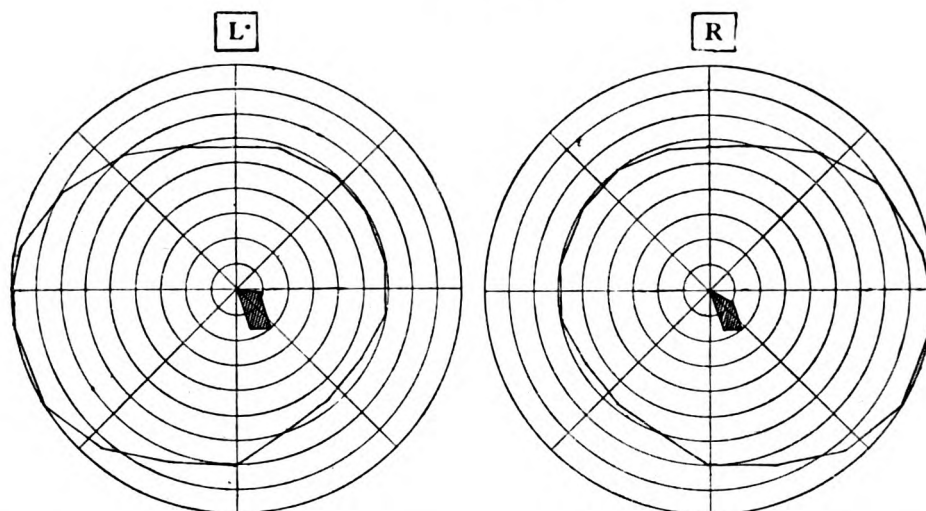


FIG. 254.—TO ILLUSTRATE THE CORTICAL LOCATION OF THE MACULAR AREA. EFFECT OF PUNCTURED WOUND OF OCCIPITAL POLE. (WILBRAND, 466.)

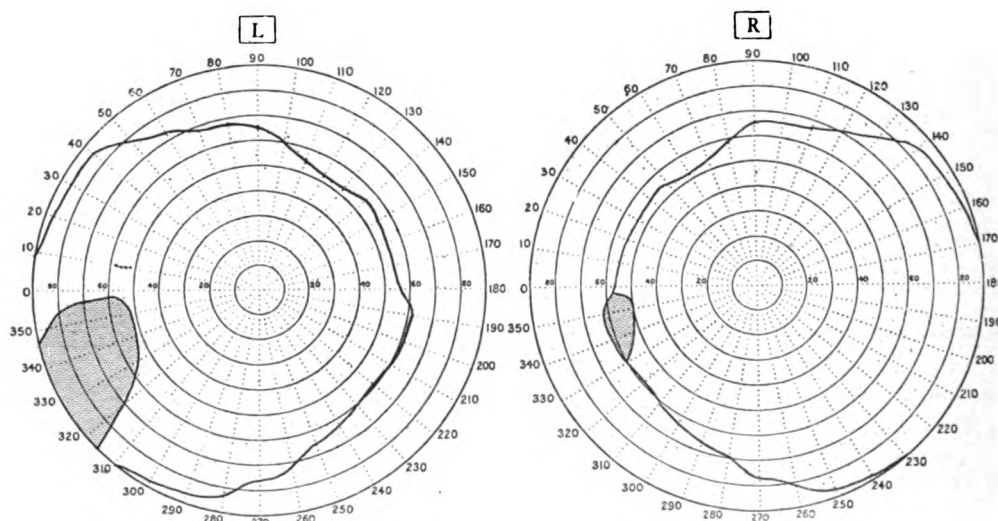


FIG. 255.—TO ILLUSTRATE THE CORTICAL LOCATION OF THE FIELD PERIPHERY. EFFECT OF PUNCTURED WOUND OF ANTERIOR LIMIT OF VISUAL CORTEX. (ADROGUÉ, 5.)

Pfeiffer holds that the cortical macular area is wedge-shaped with its base at the occipital pole and its apex extending for a considerable but varying distance forwards along the post-calcarine sulcus.

Adrogué (5) believes that central vision is represented widely throughout the visual cortex and that there is no bilateral representation.

It is believed by some observers that the most peripheral crossed fibres, *i.e.*, those from the medial periphery of the retina which correspond to the unpaired part of the temporal field, or temporal half-moon, form an isolated bundle throughout their course to the anterior part of the calcarine visual cortex. In the posterior part of the chiasma they occupy the lateral edge, beyond the uncrossed bundle, and thus theoretically enable a unilateral temporal field defect to arise from a lateral chiasmal interference. (Lenz, 243.)

According to Pfeiffer these fibres form the inferior part of the optic radiation and curve forwards into the temporal lobe as the temporal loop of Meyer.

Brouwer and Zeeman have found in apes that in the lateral geniculate body the upper retinal neurones lie medially and the lower ones laterally. The macular neurones occupy a relatively large area interposed dorsally between the peripheral retinal neurones. The neurones from the medial edge of the retina, corresponding to the unpaired area of the temporal field, occupy the lower margin of the geniculate body. The crossed and uncrossed fibres lie close together.

There is still much difference of opinion among authorities on the whole question of the visual path, and the subject is too large and too complicated for discussion here. (See Adrogué 5, Lenz 244, Henschen 168-170, Bunge, Ask-Upmark, Putnam, Malbran, Le Gros Clark and others.)

IV. Sheath and Connective Tissue Framework of the Optic Nerve

The optic nerve has dural, arachnoid, and pial sheaths. The dural sheath is continued forwards from the dura mater and blends with the sclera. The arachnoid is extremely thin and lies immediately beneath the dura. The pial sheath closely invests the nerve and gives off septa between the fibre bundles. The nerve lies loosely in the dural and arachnoid sheaths in the orbit, but is tightly embraced by them in the optic canal, but not so closely that fluid cannot pass along the sheath from the cranial cavity.

A system of trabeculae derived from the pial sheath divides the fibres into numerous small bundles and carries in vessels, exercising in this way a nutritive as well as a mechanical function. This framework is



FIG. 256.—THE CONNECTIVE TISSUE SEPTUM OF THE INTRACRANIAL PORTION OF THE OPTIC NERVE. (WILBRAND AND SAENGER, 469, VOL. VI.)

especially dense in the most vascular parts of the nerve, the anterior orbital and the foraminal portions. In the intracranial portion the arrangement of the trabeculae changes and towards the chiasma a well-marked septum appears, passing obliquely from the upper surface of the nerve downwards and inwards to or somewhat below the level of its centre dividing off a supero-medial group of bundles. This septum ceases in the anterior part of the chiasma in which the trabeculae also disappear. No trabeculae are present in the tracts.

Wilbrand and Saenger believe that this network predisposes to neuritis in the nerve as the trabeculae become thickened and compress the fibres, which play only a passive role in the inflammatory process.

A detailed description is given by Behr (28) who also draws attention to the way in which the anatomical structure of the nerve influences the pathological processes which affect it. In the bony canal the strong connection between the pial and dural sheaths is of significance in the case of fracture of the skull, since the nerve may be compressed or torn at this fixed point. Inflammation, as in syphilis, passes along the septal framework and impairs or stops nutrition and during healing cicatricial contraction may asphyxiate the nerve fibres. Where the framework is more coarsely constructed, as in the intracranial part of the nerve, in which the finer capillary carrying septa are almost altogether absent, nutrition is less efficient especially as regards the papillo-macular fibres. On this basis an explanation is offered of the Foster Kennedy syndrome, and of the scotomata of multiple sclerosis and of toxic amblyopia.

V. Blood Supply of the Visual Path

Only those features which have a bearing on field defects are referred to here.

The pigment cells, rods and cones, and outer nuclear layer of the retina are nourished by the choroidal vessels which are quite separate from the retinal vessels except where the two sets anastomose in the neighbourhood of the optic disc. The meshes of the chorio-capillaris are especially close and regular in the macular area to which, according to Hepburn, a special arterial twig is supplied. The posterior half of the choroid is supplied by the short ciliary arteries, the anterior half by the recurrent branches of the long

posterior and the anterior ciliary arteries. Posteriorly around the optic nerve entrance and at the anterior peripheral part the arterial twigs anastomose in the choroid, but the intermediate zone is practically free from anastomoses. It has been inferred from this that in certain pathological conditions the circulation in the intermediate zone is more liable to failure than that in the anterior or posterior portion, a view which forms the basis of one of the suggested explanations of the ring scotoma in retinitis pigmentosa.

The retinal artery and vein emerge from the centre of the physiological cup in the optic disc, where the artery usually bifurcates, though it may emerge as two or more branches. The superior and inferior branches divide on or near the edge of the disc at its upper and lower poles leaving the sides, especially the lateral side, free. The course of the arteries follows that of the nerve fibres, the superior and inferior temporal branches arching over and under the macula and sending twigs downwards and upwards towards it while the nasal branches run radially towards the ora serrata. The arrangement of the vessels around the macula is best seen by studying one's own macular area in the dark room by means of a small electric lamp applied to the lower eyelid. By this means minute vessels can be seen approaching the macula much more closely than can be detected by ophthalmoscopic inspection. The boundary or watershed between the

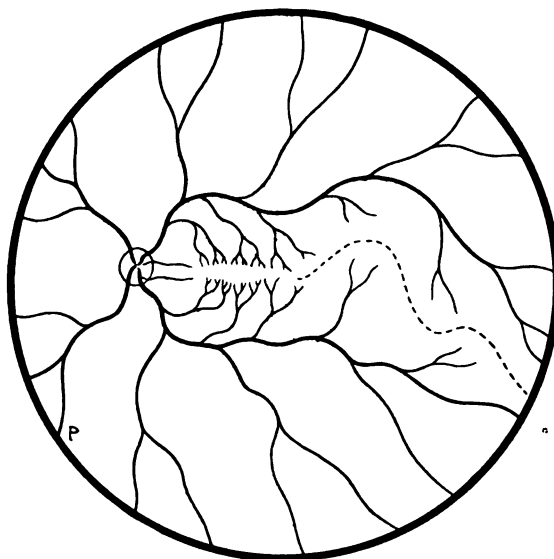


FIG. 257.—DIAGRAMMATIC REPRESENTATION OF THE VASCULAR RAPHE OF THE RETINAL ARTERY.

areas supplied by the superior and inferior temporal branches forms a fairly definite straight horizontal line in the macular area but towards the temporal part of the retina the line is wavy and only approximately horizontal. The macular area appears as an horizontally elongated and vertically flattened oval containing only very minute terminal vascular twigs which do not cross the horizontal meridian. On the lateral side of the disc small vessels extend temporally towards the medial end of the macular area. A small cilio-retinal macular twig may pass from the temporal side of the disc towards the macula.

Minute twigs from the arterial trunks on the papilla are distributed to its surface * and to the neighbouring retina and small branches on the temporal side, which pass towards the macula, often emerge separately. The circumpapillary zone may also receive a collateral supply from ciliary vessels. Sometimes a cilioretinal artery, derived, as a rule, from the scleral ciliary vessels, appears at the temporal edge of the papilla, to which it gives twigs, and passes towards the macular area. The arterial branches lie in the nerve fibre layer of the retina and give off twigs which form two capillary networks, one in the fibre and ganglion cell layers, the second in the inner nuclear layer. These are connected by intervening capillaries and the inner network is connected with the arteries, the outer with the veins (Quain). As a rule no vessels penetrate to the outer molecular layer. Lymph spaces free from capillaries surround the larger vessels. At the ora serrata the capillaries are wider than elsewhere and the network simpler so that there is almost a direct

* In obstruction of the central artery pallor of the disc occurs immediately indicating that any ciliary anastomosis in connection with the disc has little effect in maintaining or restoring its vascularity. In certain cases, in which the pressure in the central retinal artery is low, pressure on the eyeball may stop the blood current in the capillaries, and cause the optic disc to become completely dead-white in colour.

flow from the arteries into the veins. The retinal capillary network is especially rich in the macular area where the thickened ganglion-cell layer is particularly vascular. At the fovea itself there is a gap in the network about $\frac{1}{2}$ mm. in diameter, corresponding to the area in which the inner retinal layers are absent, so that the foveal cones, nourished by the choroid, are directly exposed to the light, their ganglion cells, dependent on the retinal system, being, as it were, pushed aside together with their capillaries. The superior and inferior arterial twigs which supply this perifoveal capillary network participate approximately equally so that a vascular raphé, already referred to, extends from the temporal periphery to the optic disc in a somewhat undulating course, straight in the macular area which it divides horizontally.

According to Salus (360) the fovea, or rather foveola, is not always devoid of capillaries, and the outer layers of the retina receive their nourishment from the retinal artery and not from the choroid.

The retinal arteries are all end arteries without anastomosis and there is no collateral supply excepting immediately around the optic papilla. Thus the periphery of the vascular distribution corresponds to that of the nerve distribution (Figs. 28, 257). Complete obstruction leads to ischaemic necrosis of the nerve fibre and ganglion cell layers in the area of the blocked branch though the outer layers remain nourished by the choroid. It is important to bear the vascular periphery in mind in connection with the attribution of field changes, such as those caused by quinine or tobacco, to vascular causes.

The orbital portion of the optic nerve is supplied by branches of the ophthalmic artery which leave the trunk while it is still within the optic foramen or at the anterior end of the foramen. These may be divided into two groups :—

The anterior optic nerve arteries enter the nerve sheath along with the central artery of the retina. Some of these are distributed in the pial sheath whence finer twigs penetrate along the septa. Others pass into the centre of the nerve and supply the septa from the centre outwards. As a rule a relatively large branch passes a short distance backwards in the centre of the nerve at the point where the central artery turns forwards. The region of the lamina cribrosa is especially vascular. In front of the entrance of the central artery no vessels penetrate the dural sheath.

The posterior arteries of the optic nerve, about twenty in number, pass individually through the dural sheath on all sides and are distributed to the middle and especially the posterior thirds of the nerve.

The optic nerve is entirely supplied by these branches passing through the sheath. All twigs of the ophthalmic artery or of the central retinal artery which supply the optic nerve are end arteries after they have entered the nerve. Any anastomosis there is between them takes place in the sheaths of the nerve before they enter its substance.

The veins are distributed similarly to the arteries with the exception of the central vein which receives numerous branches from the anterior and middle thirds of the nerve.

The *arteria centralis retinae* is a branch of the ophthalmic or of a ciliary artery and leaves the ophthalmic trunk at the level of the anterior end of the optic foramen together with small branches destined for the optic nerve. It enters the dural sheath of the nerve in its lower medial quadrant 10 to 15 mm. behind the eyeball. After lying in the subarachnoid space for a short distance it passes into the centre of the nerve nearly at a right angle and runs forwards to the retina thus having four bends in its course at this part. It does not pierce the pial sheath but carries a prolongation of it into the nerve. Before entering the nerve it gives off twigs to the nerve, but when it has entered it is entirely a retinal artery and gives off no branches and does not contribute to the nourishment of the nerve until it has reached the region of the lamina cribrosa, when it gives off a few branches which form a capillary anastomosis with twigs from the circle of Zinn. (Wolff, 474, 475 ; Igersheimer, 203.)

The central vein, on the other hand, collects many branches from the nerve as well as from the retina and the region of the lamina cribrosa. It emerges usually somewhat nearer the eyeball than the artery and lies for a short distance between the sheaths before piercing the dura. It passes directly to the cavernous sinus or opens into the superior ophthalmic vein. In the foraminal portion the axis of the nerve is again occupied by a small vein, which helps to drain the orbital portion and, becoming superficial in the foramen, passes backwards to the cavernous sinus. (Behr 28, Damel, 76.)

According to Beauvieux and Ristich the central artery gives twigs to the pial sheath of the nerve in the lower portion of its orbital part, but gives no branches in the region of the lamina cribrosa, which is entirely supplied by the ciliary arteries. The central artery does not anastomose anywhere with arterial branches from other sources.

There is a connection through the superior orbital fissure between the lacrimal artery and the middle meningeal, and on this account obstruction of the ophthalmic artery or of the internal carotid does not necessarily produce the appearance of obstruction of the central artery of the retina.

The central artery of the retina or the ophthalmic artery itself may come from the middle meningeal. Connections with vessels of the opposite side through the supraorbital, dorsal nasal and other branches,

also exist so that the internal carotid may be tied both above and below the origin of the ophthalmic artery without affecting the central artery of the retina. (See Walsh-King, 457.)

The chiasma receives branches from the internal carotid, anterior cerebral, and anterior and posterior communicating arteries which supply each side as far as the median plane. The pial covering is slight except at the lower and posterior part where the dense vascular network which surrounds the infundibulum occupies the angle between the two structures.

The anterior end of the tract, immediately posterior to the chiasma, is surrounded by a rich anastomosis of fine branches of the anterior choroid, internal carotid, anterior cerebral and posterior communicating arteries. The remainder, up to the lateral geniculate body, receives branches from the anterior choroid artery only. These vessels pass to the medial side of the tract, between it and the peduncle, and enfold the tract which they enter on its superior surface. Except at the anterior end of the tract there is no pial anastomosis.

Owing to the arrangement of the vessels it seems probable that pressure on the surface of the optic nerve, chiasma, or tract may disturb the nutrition of the underlying fibres by obstructing their vascular supply, and that conduction interference and ultimately optic atrophy may be initiated in this way, rather than by the effects of pressure on the fibres themselves.

The anterior and lateral aspects of the lateral geniculate body are nourished by the anterior choroid artery; the medial, posterior, and most of the central regions of the nucleus receive their blood supply from the posterior cerebral artery. Branches of the two arteries anastomose on the surface of the nucleus. The intermediate or macular segment of the nucleus has the richest blood supply, which is drawn from both arteries. This arrangement favours sparing of the macular projection in cases of arterial obstruction. (Abbie 1, q.v. for details.)

Over the geniculate body the entering vessels are more numerous and the pia mater more closely attached than over the tract.

Practically the whole of the posterior limb of the internal capsule, including the commencement of the optic radiation, is nourished by the anterior choroid artery: the main supply of the optic radiation is from the posterior cerebral artery, especially its calcarine branch. In the most posterior part the visual white matter also receives penetrating twigs from the middle cerebral.

The visual cortex obtains its blood from the calcarine artery, and, at the occipital pole, from the middle cerebral also so that the macular centre lies on a "frontier zone," between the territories of the posterior and middle cerebrals. Thus the macular area may remain unaffected in obstruction of the calcarine artery and sparing of the fixation area may result. The terminal branches of these two vessels anastomose in the pia mater, where they form a rich arterial network which sends in twigs which are end arteries. These twigs are of two kinds, the long and the short, the former pass to the subcortical nerve fibres, the latter to the cortical layers, so that the nutrition of the grey matter is independent of that of the white. It is, therefore, possible for small isolated softenings affecting the subcortical white matter only to occur (Henschen). Charpy, in Poirier's "Anatomy", points out that all cerebral arteries, having once entered the brain substance, are end arteries, but the importance of the territorial surface distribution is diminished by the presence of an anastomotic network in the pia mater covering the convolutions. This network is much less vascular over the white matter at the base of the brain.

Similarly the veins are *apparently* end-veins, while they are in the brain substance, and do not anastomose. They open into the pial venous network whence the efferent veins, which communicate freely, pass to the various sinuses. Hence, thrombosis of the veins, if it occurs, would not be likely to produce the same results as arterial obstruction unless the thrombosis were sub-pial.

VI. Uses of the Perimeter and Screen otherwise than in Field Testing

In addition to the examination of the visual field, the perimeter can be used for other purposes, which are merely enumerated here, as they do not properly fall within the scope of this work.

The perimeter may be used for the estimation of the angle of squint, and the angle between the visual and optic axes.

The presence and amount of exophthalmos may be ascertained.

In paralysis of ocular muscles the field of fixation may be measured on the perimeter and the diplopia may be studied on the screen.

The position of the tear or hole in detachment of the retina may be determined.

VII. Tables for Charting from Bjerrum's Screen

TABLE I

Tangents for the construction of tangent scales for use with Bjerrum's screen. (Maddox, 275.)

Degrees.	Tangents.	Degrees.	Tangents.
1	0.01745	23	0.4245
2	0.03492	24	0.4452
3	0.0524	25	0.4663
4	0.0699	26	0.4877
5	0.0874	27	0.5095
6	0.1051	28	0.5317
7	0.1228	29	0.5543
8	0.1405	30	0.5773
9	0.1584	31	0.6009
10	0.1763	32	0.6249
11	0.1944	33	0.6494
12	0.2125	34	0.6745
13	0.2309	35	0.7002
14	0.2493	36	0.7265
15	0.2679	37	0.7535
16	0.2867	38	0.7813
17	0.3057	39	0.8098
18	0.3249	40	0.8390
19	0.3443	41	0.8693
20	0.3640	42	0.9004
21	0.3839	43	0.9325
22	0.4040	44	0.9657

To use the table multiply the distance of the patient from the screen in millimetres (radius) by the tangent. Thus, if the screen is used at 2,000 mm., 1° will be $0.01745 \times 2,000$ mm. from the fixation point, and so on.

TABLE II

The values of successive concentric circles at intervals of 10 cm. round the fixation point on the screen. (Roenne, 336.)

Distance from fixation point. Centimetres.	Screen at 1,000 mm.	Screen at 2,000 mm.
10	$5^\circ 43'$	$2^\circ 52'$
20	$11^\circ 19'$	$5^\circ 43'$
30	$16^\circ 42'$	$8^\circ 32'$
40	$21^\circ 49'$	$11^\circ 19'$
50	$26^\circ 34'$	$14^\circ 02'$
60	$30^\circ 58'$	$16^\circ 42'$
70	$34^\circ 59'$	$19^\circ 17'$
80	$38^\circ 40'$	$21^\circ 49'$
90	$41^\circ 59'$	$24^\circ 14'$
100	45°	$26^\circ 34'$
110	$47^\circ 43'$	$28^\circ 49'$
120	$50^\circ 21'$	$30^\circ 58'$

The values for distances of 1,200, 2,400 and 2,270 mm. are also given in Roenne's paper.

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